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Acute and Chronic Traumatic Aneurysms of the Thoracic Aorta with Emphasis on Roentgenology

A Study of Sixty Eight Patients

ACUTE AND CHRONIC TRAUMATIC ANEURYSMS OF THE THORACIC AORTA WITH EMPHASIS ON ROENTGENOLOGY

A STUDY OF SIXTY EIGHT PATIENTS

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To the memory of my parents
To Marie-José, Susanne en Marjolein

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Chapter 1

INTRODUCTION AND OUTLINE OF THE STUDY

The essential ingredient of diagnosis of traumatic aortic rupture is a high index of suspicion (McBurney and Vaughan, 1961).

INTRODUCTION

The era of high speed automobile transportation with a subsequent trauma has increased the incidence of thoracic aortic rupture (TAR). Between 1936 and 1942 Strassmann (1947) in a series of 7000 autopies found only 72 patients with traumatic rupture of the aorta secondary to vehicular collision. Greendyke (1966), in the USA, reported a 16% incidence of aortic ruptures in victims of fatal automobile accidents and Zeldenrust and Aarts (1962) reported autopsy data of 88 patients with aortic rupture in 800 traffic accidents with fatal outcome in The Netherlands. Though the acute traumatic aortic rupture is usually rapidly fatal, 15 to 20% of the patients reach a hospital alive. Initial survival is dependent upon containment of the rupture within the aortic adventitia and/or the pleural tissue. A traumatic laceration of the aortic wall is always an unstable lesion and therefore the short term prognosis of patients reaching the hospital alive is poor if no treatment is started: 25 to 30% will die within 24 hours, 90 to 95% within 3 months (Parmley et al., 1958). Long term survival of untreated cases with formation of a chronic pseudo-aneurysm is unusual and occurs in only 2 to 5% of the reported cases (Parmley et al., 1958; McBurney and Vaughan, 1961).

Adequate and speedy diagnosis is needed for patients with acute aortic rupture because operative treatment and complete healing is now possible; survival rates of even 70 to 90% are reported in these multi-trauma patients, who often have a combination of thoracic, cerebral, abdominal and extremity injuries (Kirsh et al., 1976;Turney et al., 1976; Vasko et al., 1977).

The diagnosis of acute aortic rupture is often difficult (Appelbaum et al., 1975).

The clinical picture of aortic rupture is aspecific with a paucity of specific symptoms; attention is often focussed on the concomitant injuries. The diagnosis therefore depends mainly on a history of a deceleration trauma and the radiological evaluation of the plain chest roentgenogram, especially of the mediastinum.

In a clinical setting, 95% of all traumatic aortic ruptures occur in the region of the isthmus (Symbas, 1972). The trauma causes disruption of intima and media, and a local false aneurysm develops contained by the adventitia and/or the mediastinal pleura. In case of aortic rupture a mediastinal hematoma is nearly always present, due to either concomitant bleeding of small arteries and veins in the mediastinum or

subadventitial or paraaortic leakage of blood. As a consequence of the mediastinal hematoma and/or the false aneurysm, changes occur in the contours of the mediastinum. Recognition of these abnormal contours on the chest X-ray is therefore essential for suspecting the diagnosis of aortic rupture.
In trauma patients the mediastinum is not easy to evaluate because chest films have usually to be obtained in a supine position, at short focus-film distance.
In addition, mediastinal hemorrhage can occur without aortic rupture as bleeding may originate from small arteries and veins in the mediastinum. Although there are many reports on radiological evaluation of the mediastinum in patients with TAR in the literature, most series except some recent ones (Appelbaum et al., 1975; Kirsh et al., 1976; Vasko et al., 1977; Richardson et al., 1979; Fisher et al., 1981; Barcia and Livoni, 1983) are restricted. In 1958 the first successful operation for an acute traumatic aortic rupture was performed by Klassen (Passaro and Pace, 1959). From then on the prognosis of patients with an aortic rupture was improved. If correct clinical and radiological evaluation is followed by an operation, the survival rate can be 60-90% (Kirsh et al., 1970; 1976; Turney et al., 1976; Vasko et al., 1977).
Patients with an acute aortic rupture who reach the hospital alive are usually in an unstable condition. A major risk is fatal rupture before diagnosis and subsequent treatment are possible. Because these patients are often multi-trauma patients, the other injuries will influence the ultimate prognosis. Sometimes it is even necessary to treat the other injuries before treatment of the rupture (Kirsh et al., 1976; Turney et al., 1976; Bodily et al., 1977).

OUTLINE OF THE STUDY

After a review of the literature of TAR, we will discuss our own patient data.
Between 1970 and 1984 we have seen 44 patients with an aortic rupture in the University Hospital St. Radboud in Nijmegen, 35 exhibited an acute traumatic aortic rupture, 9 a chronic post traumatic aortic aneurysm (CTAR). We were also able to review the data of the 24 patients seen with an acute rupture in the University Hospital in Groningen between 1976 and 1984, 22 patients with an acute traumatic aortic rupture, and 2 with a chronic post-traumatic aneurysm. We present the clinical and radiological findings of these 68 patients. The radiological findings on the chest X-ray both in the acute and chronic group will be reviewed in detail, and frequency of occurrence of the different signs on the chest X-ray will be presented. We compare these radiographic findings in the patients with TAR with those of a group of 62 blunt chest trauma patients who had a negative thoracic angiogram. By analysing and comparing the chest X-ray findings of both groups we will determine the significance and the predictive value of each of these findings or of a combination of these in order to establish more definite radiological criteria for the suspicion of aortic rupture. The (future) role of new imaging methods like digital

subtraction angiography (DSA) and computer tomography (CT) in diagnosing aortic rupture will be discussed. We will show the different positive signs of TAR on the chest roentgenograms and angiographies of patients with TAR. Furthermore, we analyse the major factors influencing the outcome of our 57 patients with acute TAR. Frequency and significance of other traumatic injuries in the patients with TAR will be discussed. We review some aspects of surgery for TAR both in the literature and in the own patients series.

We describe and review the clinical findings and chest roentgenograms on primary and secondary admission of 11 patients with a chronic traumatic aneurysm (CTAR). We will try to analyse, why the diagnosis of TAR at the first admission was missed.

In short, the aim of this thesis is:

to review the literature on TAR

to discuss the role of new radiological techniques (DSA, CT) in diagnosing TAR (chapter II),

to present 57 patients with acute and 11 with chronic TAR,

to evaluate radiological findings on the chest-X ray in order to establish more definite radiological criteria for the suspicion of acute aortic rupture (chapter III),

to analyse the influence of concomitant trauma and discuss some aspects of surgery (chapter IV and V),

to analyse factors influencing prognosis of patients with TAR (chapter VI),

to discuss chronic traumatic aneurysms (chapter VII and VIII).

Part I

ACUTE TRAUMATIC ANEURYSMS OF THE THORACIC AORTA

GENERAL ASPECTS OF TRAUMATIC ANEURYSMS OF THE THORACIC AORTA (survey of the literature)

II.1. **History**

Prior to the time of rapid transportation, traumatic aortic injury by blunt chest trauma was rare.

Vesalius, in 1557, first reported a thoracic aortic lesion from blunt trauma in a man who developed a pulsatile tumor of his back, many years after falling from a horse. Until 1947, there were sporadic reports on traumatic aortic rupture in the literature. Copeland (1914) reported a traumatic aneurysm of the ascending aorta caused by outside compression. Oppenheim's (1918) case was a blast injury causing a traumatic aneurysm of the ascending aorta. In 1919 Jaffé and Sternberg reported 10 cases of traumatic aortic rupture at the isthmus, five of which were aircraft injuries (World War I!) and five were crash- and deceleration injuries. Samson (1931) stated after observing two cases of probable traumatic aneurysm: 'traumatic aneurysms of the aorta are practically all dissecting'. Hawkes (1935) performed 7000 autopies and found 16 cases of traumatic aortic rupture. Case reports of one or two cases (Collins and Alesio, 1938; Kleinsasser, 1943; Leonard, 1945; McDonald and Campbell, 1945) were followed by the classical report of Strassmann (1947) with 75 cases, seen between 1936 and 1942, in 7000 autopies in New York. Two-thirds of the cases were caused by automobile accidents. In 1958 Parmley et al. published the largest for the present, series of 275 cases from the Armed Forces Institute of Pathology. All of the above mentioned cases, except two in Parmley's series with a chronic traumatic aneurysm, were found at autopsy. The rising number of reported cases parallels the higher frequency of automobile accidents (Greendyke, 1966).

The first operations for a chronic traumatic aortic aneurysm were done by Weisel (1951), Hollingsworth et al. (1952), Bahnson (1952) and Stranahan et al.(1953). In 1959 Klassen (reported by Passaro and Pace) operated successfully for the first time upon a patient with an acute traumatic aneurysm, crossclamping the aorta for 17 minutes and suturing a 3 mm tear at the aortic isthmus. Cooley et al. (1957) were the first to resect a chronic traumatic aneurysm and place a graft. They used hypothermia to protect the distal circulation. In 1957 Cooley et al. and Gerbode et al. introduced left atrial to femoral artery bypass to prevent distal ischemia and left ventricle strain when crossclamping the aorta. Blake et al. (1960) were the first to employ angiography in the pre-operative diagnosis of acute aortic rupture by contrast injection in the right atrium. Jahnke et al.(1964) used as first retrograde angiography via the femoral route.

Table II.1 Angiographic series of TAR

Year	*Author*	*Nr. of patients with TAR*
1971	DeMeules et al.	15
1975	Appelbaum et al.	25
1976	Kirsh et al.	38
1977	Vasko et al.	17
1980	Motin et al.	36
1981	Akins et al.	44
1981	Fisher et al	47
1983	Barcia and Livoni	17

In The Netherlands, Zeldenrust and Aarts in 1962 focussed attention on acute aortic rupture, reporting 88 cases in 800 lethal traffic accidents. Their cases were diagnosed at autopsy. Lacquet et al.(1972, 1973, 1974, 1975, 1978), Van Lent et al. (1972), Skotnicki et al. (1976, 1982), Brutel la Rivière et al. (1981) and van Eggermond and Hiemstra (1981) reported surgical series of patients with an acute aortic rupture: most of them were operated upon with success. Puylaert (1976) reported several cases with emphasis on radiological findings. However, few of them survived to be operated upon. Relatively large angiographic series of patients with acute aortic rupture have been reported since 1971 (table II.1).
Only the reports of Fisher et al.(1981) and Barcia and Livoni (1983) analyse in detail the radiological findings in patients with an acute aortic rupture.

II.2. **Frequency of occurrence**

Before the era of high speed automobiles acute traumatic rupture of the aorta (TAR) was rare. Autopsy data from the literature are tabulated in table II.2.

Table II.2 Autopsy data and TAR

Year	*Author*	*Nr. autopsies*	*Nr. TAR*
1935	Hawkes	7000	16
1941	Ruffin et al.	9600	0
1947	Strassmann	7000	72
1958	Parmley et al.	1174	275
1962	Zeldenrust and Aarts	800	88
1963	Lundevall	1200	20
1971	Heberer	389	160

Hawkes (1935) reported 16 cases of TAR in 7000 autopsies, Ruffin et al.(1941) were unable to attribute to trauma any of the 66 thoracic aortic aneurysms in a review of 9600 autopsies at the Massachusetts General Hospital. Strassmann (1947) published 72 cases of TAR in 7000 autopsies performed between 1936 and 1942 in the New York City Medical Examiners Office and in 1958 Parmley et al. reported 275 cases from the files of the Armed Forces Institute of Pathology. These 275 cases were found at autopsy in 1174 cases of traumatic injury to the heart and the aorta. Lundevall (1971) reported 20 cases in a ten year study involving 1200 medico-legal autopsies at the Institute of Legal Medicine in Oslo between 1953 and 1962. Heberer (1971) reported on 389 patients from the Cologne Institute of Forensic Medicine with blunt chest trauma between 1965 and 1970. In 160 cases aortic injuries were found, which were lethal in 99 cases. Keen (1972) published from the Bristol area (600.000 inhabitants) about the 18 survivors and 19 autopsies with TAR in a six year period. The only data for The Netherlands are available from the report of Zeldenrust and Aarts (1962). They found 88 aortic ruptures at autopsy in 800 fatal automobile victims.

From epidemiological experience, the following data are available. On reviewing 194 patients with chest injuries in car accidents, Molnar and Pace (1966) found that 25% sustained major injuries of thoracic organs and 3.5% had TAR. In 1969, 56.000 fatal automobile accidents occurred in the USA. According to the estimates of Greendyke (1966) and Zeldenrust and Aarts (1962), TAR accounts for 10 to 15% of traffic fatalities. This means that there were between 5600 and 8000 cases of fatal TAR in the USA in 1969. Wilson et al. (1972) stated: 'it has been estimated that about 7000 automobile accidents occur each year in the USA which are associated with TAR. Approximately 10-20 percent of these victims live long enough to be brought alive to an emergency department.' Bodily et al.(1977) estimated that there were approximately 1000 potential survivors of TAR in the USA each year. The incidence of reported cases rises sharply in patients at the age of 17, is the highest at the age of 20, and declines thereafter but continues throughout the life expectancy. Meyer et al.(1969) described an 8 year old boy with TAR.

It is known from the studies of Greendyke (1966) and Parmley et al. (1958) that TAR occurs in 15 to 20 percent of autopsied automobile victims. This would mean for The Netherlands with approximately 2000 fatal traffic accidents each year 300 to 400 cases of TAR. Of these victims 15-20% reach the hospital alive, giving about 40-80 clinical cases. In the files of the 'Stichting Medische Registratie' respectively 28, 21, 17 cases of TAR were registered in the years 1980, 1981, and 1982. These figures make underdiagnosis of TAR in our country probable. From data of the CBS (Central Dutch Institution for Statistics) there is a sharp decline in the number of traffic deaths from 1970-1981 (1970: 3181; 1975: 2321; 1981: 1807 deaths). Two-third of the deaths were due to automobile accidents.

II.3. **Ethiology and pathogenesis of aortic rupture**

The majority of TAR is due to car- or motorcyle accidents where acute horizontal deceleration and impact takes place. Table 11.3 summarises some reports from the literature.

Table II.3 Percentage of car- or motorcycle accidents as cause of TAR

Year	*Author*	*Country*	*%*
1947	Strassmann	USA	61%
1958	Parmley et al.	USA	56%
1962	Zeldenrust and Aarts	The Netherlands	66%
1963	Lundevall	Norway	95%
1964	Jensen	Danmark	87%
1966	Greendyke	USA	83%
1969	Rittenhouse et al.	USA	76%
1974	Fleming and Green	USA	90%

However, tears have been found in victims of air plane crashes (Jaffé and Sternberg, 1919; Hass, 1944; Wilson, 1946; Teare, 1951; Jackson and Mazur, 1965; Cocket, 1981), glider crash (Skotnicki et al., 1982), fall from a height (Strassmann, 1947; Parmley et al., 1958); following free fall of lifts to the ground (Strassmann, 1947; Parmley et al., 1958); after direct blows on the chest not associated with body deceleration (Kemp, 1923; Strassmann, 1947; Parmley et al., 1958); after a blow on the epigastrium (Forbes, 1944); as a result of severe blast injury (Oppenheim, 1919; Kemp, 1923; Wilson, 1943); after compression (Parmley et al., 1958). Typical for The Netherlands is a bicycle-car collision (Skotnicki et al., 1982). Although much has been written, the factors responsible for traumatic rupture of the thoracic aorta have not been clearly defined. Different mechanisms are discussed in the literature.

A. *Congenital - anatomic factors*

Abbott (1928) has been falsely credited with stating that the isthmus is congenitally weak. In actuality, however, her reference pertained only to the ascending aorta in patients with a bicuspid aortic valve and to the descending aorta in patients with coarctation of the aorta. Lundevall (1964), using cadaver aortas, demonstrated less resistance to stretching in the aortic isthmus compared with the ascending and descending aorta. He also showed less tensile strength in all segments of the aorta to longitudinal stress rather than transverse stress; this correlates well with the clinical and autopsy findings that the overwhelming number of aortic ruptures are transverse. An additional important anatomic consideration lies in the tensile strength of the

aortic wall. Whereas distension below 100 mmHg is resisted mainly by the media, higher pressures are resisted by the adventitia, which possesses greater tensile strength (Sanborn et al., 1960). The presence of the strong adventitia, which provides 60% of aortic wall strength permits survival after aortic rupture (Eiseman and Rainer, 1958; Butcher, 1960).

B. *Mechanico-physical factors*

Rindfleisch (1893) held the opinion that traumatic rupture of the aorta is produced by a sudden stretching of the vessel wall and that the upper thoracic portion of the aorta is most exposed to such a stretching. Oppenheim (1918) ligated the branches of human aortas and filled the aortas with water reaching a pressure of 3000 mmHg. In healthy aortas rupture occurred above the valves and at the isthmus at 2700 mmHg. Dissecting aneurysms frequently originated during the experiments. Klotz and Simpson (1932) found in similar experiments that the aortas from young persons resisted an internal pressure of 1000 mmHg without rupturing, but they nevertheless assumed that it is the sudden rise of blood pressure by which the aorta ruptures during accidents. Kleinsasser (1943), Strassmann (1947) and Tannenbaum and Ferguson (1948) shared the opinion that an acute rise in blood pressure is an important cause of aortic rupture.

According to Hass (1944), different rates of deceleration of body parts evoke stress at the connection of these parts. He stated: 'this stress is proportional to the rate of deceleration. In high speed deceleration injuries the central portion of the descending aorta is snapped forward by the momentum of the deceleration force and the mass of the aortic blood content. The arch of the aorta, which is fixed by the great vessels, decelerates at the same rate as the body. Because these two parts of the aorta decelerate at a different rate, the greater stress is placed upon the aortic isthmus'.

Zehnder (1956) deduced from theoretical considerations that the main factor in different traumata is a hyperflexion of the aortic arch, with a predominant straining stress at the isthmus region. Cammack et al.(1959) stated that anterior-posterior compression of the chest wall causes dislocation of the heart to the left and posteriorly, producing torsion and shearing stress on the aortic wall with maximum stress near the base of the heart and the isthmus region.

Flaherty et al.(1959) held the same opinion as Hass, stating that the aortic fracture is caused by the straining force generated by differences in inertia of relative mobile and relative immobile segments of the aorta. Lundevall (1964) studied the aortas of 10 specimens and found the thoracic part of the aorta relatively mobile but the isthmus region slightly more fixed to its surroundings. The great branches from the aortic arch did not particularly arrest the movement of the aorta nor did the intercostal arteries. The root of the left lung was relatively fixed. He stretched the aortic wall (after removing the adventitia) until rupture and found resistance to rupture reduced at the isthmus.

He concluded from his experiments that three kinds of mechanical factors may act on the aortic wall during rapid velocity changes:

1. traction and pressure on the aortic wall by other thoracic organs and torsion of the aorta
2. strain waves in the aortic wall itself
3. pressure variations of the aortic blood content.

Jackson et al.(1968) experimented with blows on the chest of dogs and subsequent compression of the thoracic cage. They noticed that the sternum was displaced posteriorly to approximate the vertebral column. The aorta moved laterally with increase in length. This resulted in a disrupting force applied in cranio-caudal direction and explained the transverse nature of the tears. Acute ruptures have sites of predilection and an explanation of the ethiology of rupture must take this into account. The vulnerability of special sites does not favour direct trauma to the aorta as a mechanism. Moreover, the deep location of the proximal descending aorta would seem to protect it from rupture by anterior blows on the chest. Furthermore, chest trauma is absent in some cases. Anatomically fixed points are relevant. The aorta is a distensible tube, which will also stretch longitudinally but is relatively nonelastic. Like a overstretched rubber tube it has the potential of transverse tearing when sufficient longitudinal tension is applied. This seems likely to be one of the final modes of rupture, possibly the common final pathway to tearing, whatever the precipating forces are (Sevitt, 1977).

Gotzen et al.(1980) held the opinion that TAR was most frequently found in car passengers not wearing seat belts; Watz et al.(1978) reported however aortic rupture in 14 patients wearing seat belts, but in all severe chest wall trauma was found as well. In the opinion of Gotzen et al.(1980), seat belt wearers do not suffer aortic rupture unless there is additional direct chest wall trauma.

The most recent discussion on ethiologic factors of aortic rupture was made by Sevitt (1977). He discussed as ethiologic factors 4 different mechanisms and compared his own findings with the literature. These mechanisms are:

1. direct blunt force to the chest
2. deceleration effects
3. a combination of direct blunt force and deceleration
4. acute intra-aortic pressure rise.

1. *Direct blunt force to the chest*

Ruptures have been found in standing subjects struck violently on the chest by heavy objects as reported by Kemp (1923) and Strassmann (1947).

Ruptures of the ascending aorta were produced by the experiments of Moffat et al.(1967) who struck dogs in the mid sternal area with an impactor. Jackson et al.(1968) produced tears of the ascending and descending aorta in an identical experiment.

Louhimo (1967) performed angiographic studies in rabbits with the same kind of trauma and stated that dislocation of the heart caudally and to the left played an important role in producing aortic ruptures. A similar explanation is offered by Voigt (1968) for ruptures in the ascending aorta in drivers after horizontally directed steering wheel impact on the chest; heart and aorta displacement were demonstrated angiographically in compression tests in cadavers. It seems reasonable to conclude that many ruptures of the ascending aorta, associated with severe blows on the chest are produced by a stretching tension following displacement of the heart downward and to the left.

Voigt (1968) stated after careful autopsy studies in trauma patients: 'in many cases of frontal collision the impact is on the lower chest wall and directed in cranial and posterior direction. Because of this impact and its direction, the mediastinum including the heart and ascending aorta are displaced cranially by the lower sternum. This cranial displacement puts a tensile stretch on the proximal descending aorta which ruptures near its most fixed point at the isthmus'.

Coerman et al. (1972) reached the same conclusion from experiments with cadavers. This mechanism is called the 'shovelling' mechanism. From the same experiments they concluded that a shock absorbing steering wheel house caused less severe blunt chest trauma.

In the opinion of Götzen et al. (1980) severe compression trauma of the thorax was the common denominator and most important factor for TAR. They stated: 'the direction of the impact and the resulting thoracic deformation appears to be of great significance in the mechanism of rupture'.

2. *Deceleration effects*

After sudden arrest of the body from rapid motion, internal organs continue to move during a fraction of a second and this can produce lesions. Discovering aortic injuries after aircraft accidents, Hass (1944) stated that whereas one part of the body is decelerated at a rate different from that of another part of the body, the connection between the two parts is placed under stress, which is proportional to the difference in the rates of deceleration. This could explain aortic rupture in patients with little or no evidence of injury to the chest. Experiments involving acceleration and deceleration without any impact have been carried out by Aldman (1962). Aldman's work on pigs subjected to rapid oscillatory movements in the long axis of the body support the idea that rapid deceleration of the body moving in a cranial or caudal direction may itself cause rupture at the classic site. Deceleration in a cranial direction invokes sudden stretching tension on the superior aorta. Tears by this mechanism are most common just proximal to the ligamentum arteriosum. Simultaneous chest compression could enhance the danger of rupture by causing heart displacement and tension on the superior aorta. Deceleration from motion in a caudal direction would explain rupture of the proximal descending aorta found in cases of suicide and subjects jumping or falling from a height (Fidler, 1949).

3. *Combination of direct blunt force and deceleration*

According to Tannenbaum and Ferguson (1948) and Keen (1972), the descending aorta below the ligamentum arteriosum tends to displace forwards after sudden arrest of forward motion. This puts tension on the ligamentum arteriosum and the aorta ruptures at the site of attachment. If this explanation is correct, tearing of intercostal arteries would be expected and these are not found at autopsy. Zehnder (1956) invoked both chest compression and a deceleration thrust to explain forward bending of the aortic arch, which he regarded as the mechanism of rupture after chest impact from high speed accidents.

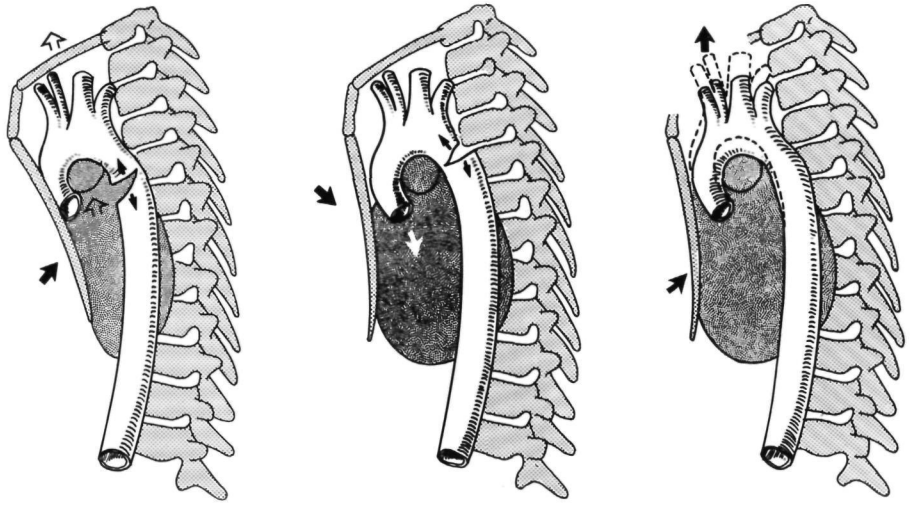
4. *Acute rise of intra-aortic pressure*

The pressure required to rupture the aorta was investigated in cadavers by Oppenheim (1918) and Klotz and Simpson (1932).Oppenheim produced ruptures at a pressure of 3000 mmHg. Klotz and Simpson produced no ruptures until a pressure of 1000 mm Hg. was reached. Lundevall (1964) regarded a sharp rise in aortic pressure (waterhammer effect) as important for traumatic rupture, but also implicated simultaneous local pressure, traction and stain waves on the wall. Moffat (1966) measured a pressure lower than 400 mm Hg. in the aortas of dogs during impact on the sternum. The overall evidence from the literature is not conclusive, but rise of aortic pressure caused by a blunt blow on the chest does not seem decisive for aortic rupture.

The conclusions of Sevitt (1977) regarding the most probable explanation for the mechanism of aortic rupture are as follows and seem reasonable: from the above data he concludes that the internal and transverse nature of all traumatic ruptures of the aorta indicates a stretching mechanism; anatomical points of fixation largely determine the location. The aortic attachment to the heart influences the supravalvular site of rupture in the ascending aorta; the attachment of the ligamentum arteriosum plays a vital part in locating ruptures in the proximal descending aorta, the isthmus. Most ruptures in surviving patients are localised in the isthmus area and can be caused in several ways:

1. by cranially directed deceleration after impact producing transient stretching of the superior aorta (Fig. II.1.a).
2. by caudally directed deceleration after displacement of thoraco-abdominal organs (Fig. II.1.b).
3. by cranially directed displacement of the mediastinum and aortic arch following a cranically directed blow on the lower part of the thorax (Fig. II.1.c).
4. possibly by horizontal directed deceleration.

More than one mechanism may be responsible in individual cases, especially in victims of automobile accidents. Most ruptures of the ascending aorta are caused by a severe blow on the front of the chest during which the heart is displaced downwards and to the left. Acute rise of aortic pressure induced by chest blows or deceleration is unlikely to be important for rupture, but is still subjudice.



1.a 1.b 1.c

Figure II.1 Mechanism of traumatic aortic rupture.
Figure II.1.a Impact on the chest wall in cranial and posterior direction produces rupture at the aortic isthmus (Modified from Götzen et al. 1981).
Figure II.1.b Impact on the chest wall in caudal and posterior direction produces rupture at the aortic isthmus (Modified from Götzen et al. 1981).
Figure II.1.c Impact on the anterior chest wall produces transient stretching of the superior part of the aortic arch and rupture of the aortic isthmus (Modified from Sevitt, 1977).

II.4. **Pathology**

The most complete description of pathology of aortic rupture is found in the review of Parmley et al.(1958). In their autopsy study, most of the aortic lesions were ruptures of the wall, varying in length from a millimeter to complete transection. They classified lesions into the following categories:

1. intimal hemorrhage
2. intimal hemorrhage with laceration
3. medial laceration
4. adventitial laceration

1. *Intimal hemorrhage*

These hemorrhagic lesions varied in size and were often only noted after microscopic examination. These lesions were sometimes found in association with other fatal injuries.

2. *Intimal lacerations with hemorrhage*

In these lesions the endothelial layer was broken and the collagenous and elastic

fibres of the subendothelial layers of the intima were more severely disrupted and separated by the hemorrhage. Lesions of this type served as the nidus for thrombus formation and dissection.

3. *Lesions of the media*

The larger part of the aortic wall is the tunica media (media). The smooth muscle fibers provide the contractile properties and serve to maintain the 'vascular tone' but probably contribute relatively little to the overall strength of the arterial wall.

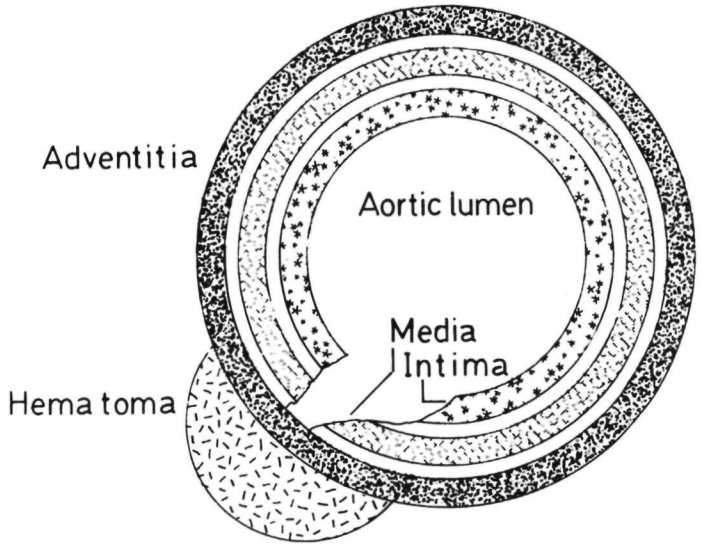


Figure II.2 Most traumatic aortic ruptures involve the intima and media, leaving the adventitia intact (Modified from Ayella et al., 1977).

Lacerations extending into, but not through the tunica media were observed occasionally in the review of Parmley et al.(1958). Laceration of the media may lead to further dissection, rupture or aneurysm formation (see dissection). Ruptures involving only part of the aortic circumference were often posterior. Occasionally ragged, spiral or longitudinal ruptures were present, but usually the rupture was transverse and smooth. About 60% of aortic ruptures usually involve the intima and media, leaving the adventitia intact (Ayella et al., 1977), (Fig. II.2.).

4. *Lesions of the adventitia*

The tunica adventitia which forms 60% of the strength of all the aortic wall layers (Sanborn et al., 1970) provides a portion of the tensile strength necessary for the maintenance of the normal caliber of the aorta and contributes through the medium

of the vasa vasorum to the sustenance of the aortic wall. Parmley et al.(1958) classified lesions involving the adventitia as:

a. traumatic false aneurysm formation
b. complete aortic laceration
c. peri-aortic hemorrhage

a. *Traumatic false aneurysm*

All the cases were included in which aneurysmal bulging of the aortic wall followed complete laceration of the intima and media and those in which a false aneurysm formed after rupture of all three layers. In those cases in which circumferential laceration of the intima and media was complete, a fusiform aneurysmal bulging of the wall of the aorta developed, while when only a portion of the wall was lacerated, the bulging appeared as a localised diverticulum or pouch-like formation. The structure varied with the age and duration of the aneurysm. In post-mortem examinations soon after the injury, the aneurysm contained a thrombus and usually there was hemorrhage into adjacent tissue. If the patient survived for a few days, fibroblastic proliferation and early signs of repair were found in the wall of the aorta.

b. *Complete laceration of the aortic wall*

A complete rupture of all three layers of the aortic wall was found in 40% of the cases. Atherosclerosis in varying degree was found in the aortas, but was considered in only one case to have added significantly to the traumatic lesion. Medial cystic necrosis of minimal to moderate degree in three cases made the aorta probably more vulnerable than usual. In the complete ruptures separation of the ends of the aortic wall often occurred, varying between 5 and 7 cm.

c. *Periaortic hemorrhage*

Periaortic hemorrhage often accompanied complete rupture or other traumatic lesions of the aorta, but frequently occurred independently. The findings of Zeldenrust and Aarts (1962) are in agreement with those of Parmley et al.(1958), on microscopic examination they often found multiple small intimal tears in the neighbourhood of a true rupture.

Sanborn et al.(1970) described radiological-pathological correlations in more than 60 patients. They stated: 'the localised, irregular fusiform widening of the aorta just distal to the arch which is seen on angiograms is the result of subadventitial extravasation of blood through a traumatic defect in the intima and media. Sometimes there is a protrusion of the transected portion of the aortic wall into the aneurysm which can be seen on the angiogram as a sharply defined defect in the contrast column at the proximal or distal margin of the aneurysm'.

Occasionally dissecting hematoma of the aortic wall occurs following chest injury (Samson, 1931; Leonard, 1945; Rice and Wittstruck, 1951; Malm an Deterling, 1960; Greendyke, 1966; Hewitt, 1970; Faraci and Westcott, 1977; Fisher et al.,

1981). The two kinds of aortic dissection -medial and subadvential which have been described following trauma, seem to depend mainly whether the aorta is normal or diseased at the time of the injury (Faraci and Westcott, 1977).
When the aorta is normal, blunt trauma results in either a laceration or transection of the intima, intima and media or all three layers. A subadventitial hematoma can develop and rupture into the mediastinum or it may compress the aortic lumen and produce an 'aorta coarctation syndrome' (Fig. II.3). This subadventitial hematoma differs from the classic medial dissection in which the hematoma is localised within the media and involves usually longer segments of the aorta. Media dissection after blunt trauma is extremely rare if the aorta is normal; Rice and Wittstruck (1951) reported media dissection in trauma in a 20 years old woman, 8 months pregnant. Other case reports of medial dissection in aortic trauma are reported by Samson (1931), Hewitt and Grablowski (1970), Katz et al.(1974), Fisher et al.(1981).
In table II.4 the localisation of the rupture in several autopsy series is summarised.

Table II.4 Localisation of aortic ruptures at autopsy

Year	*Author*	*Nr. patients*	*MI*[1].	*Asc. Ao*[2].	*Arch*	*Isthmus*	*Desc. Ao*[3].	*Abd. Ao*[4].
1947	Strassmann	72	11	14		40	15	3
1958	Parmley et al.	275	17	64	22	124	35	13
1962	Zeldenrust and Aarts	88		11		63	5	9
1966	Greendyke	42	8	4		25	5	8
1977	Sevitt	37	1	6	1	25	5	
1981	Heberer	88		11		77		

1 MI. = multiple injuries of the thoracic aorta (2 or more ruptures)
2 Asc. Ao. = ascending aorta
3 Desc. Ao. = descending aorta
4 Abd. Ao. = abdominal aorta

As can be seen from table II.4, most ruptures occur at the classical site, i.e. the aortic isthmus, although ruptures of the ascending aorta, aortic arch, descending and abdominal aorta are not infrequent. Two or more ruptures are not uncommon, the site of the second tear varies considerably (Strassmann, 1947).

II.5. **Natural history of TAR**

The natural history of patients with TAR is bad, 80-85% of the patients will die before reaching the hospital, 15-20% reach the hospital alive (Parmley et al., 1958).

Survival is dependent on the containment of the hematoma by the adventitia and/or mediastinal pleura. Prior to 1959, surgical intervention was not possible for TAR, thus the figures of Strassmann (1947) and Parmley et al. (1958) should reflect the true natural history. After 1959, the figures are influenced by surgical repair. Table II.5 summarizes the findings from the literature.

Table II.5 Mortality in TAR; natural history

Year	*Author*	*Nr. patients*	*DOA*[1]	*1 Hr*[2]	*3 Hr*	*6 Hr*	*12 Hr*	*24 Hr*	*1 Wk*[5]	*2 Wk*	*1 M*[6]
1947	Strassmann[7]	72		59	6	3		4			
1958	Parmley et al[7].	275	237	3		5		4	11	5	1
1959	Cammack et al[7].	9	4	1	1	1				1	1
1964	Jahnke et al[3].	38					7	8	5		18
1966	Greendyke et al.	42	39		2	1					
1971	DeMeules et al.	15		3			7				
1971	Wilson et al.	7					1				
1972	Keen	13		4			3		1	1	
1973	Sutorius et al.	12		1	1						
1975	Appelbaum et al.	18									
1976	Kirsh et al.	43		1	2	2					
1976	Turney et al.	31			2[4]						
1977	Bodily et al.	39	4	10	3	2	2	2	2		
1977	Vasko et al.	19									
1979	Plume and de Weese	20			4						
1981	Akins et al.	21						3			

1. DOA: dead on arrival
2. Hr: hours; time of survival after trauma
3. Review of the literature
4. Concomitant cerebral injury
5. Wk: weeks; time of survival after trauma
6. M: months; time of survival after trauma
7. Prior to the time of the possibility of operative treatment.

As can be seen from table II.5, the prognosis of a patient with TAR has improved in the recent years; early recognition is essential as can be seen from the figures of Bodily et al. (1977). Although the risk of acute death during the first hours or days in the different reports varies between 0 and 50%, one can never predict in which patient this will occur. Once the diagnosis is suspected, angiography should be performed immediately and if positive, an operation should follow as soon as possible.

The unfavourable natural history of patients with TAR depends mainly on four factors:

a. the localisation (ascending aorta versus descending aorta)
b. the concomitant injuries
c. the extent of the lesion
d. early detection and operation.

a. *The localisation*

Patients with traumatic aneurysms of the ascending aorta seldom reach the hospital alive, although rupture in the pericardial sac with tamponade seems infrequent (Strassmann, 1947). Most frequently the reason for death in these patients is concomitant laceration of the heart or other organs as the liver or the spleen (Parmley et al., 1958). In fatal isolated aortic rupture, 50 to 60% occurred in the aortic isthmus and only 10 to 20% in the ascending aorta, whereas in subjects with an associated cardiac injury, 52% of the ruptures were localised in the ascending aorta and only 32% in the region of the ligamentum arteriosum (Symbas, 1972).

Although at autopsy 20-30% of TAR is localised in the ascending aorta, 95% of ruptures are found at the isthmus at operation, reflecting the more unfavourable prognosis of ascending aortic aneurysms (Symbas, 1972).

b. *Concomitant injuries*

A prerequisite for survival of patients with TAR is the absence of concomitant lethal injuries. 20% of the 171 patients in the series of Parmley et al.(1958) with isolated TAR survived the initial injury, whereas only 4% with combined rupture and cardiac injury survived for a short time span. Greendyke (1966) published the results of 42 autopsies of patients with TAR. Two-thirds had concomitant lethal injuries. Bodily et al.(1977) in a clinical series, reported lethal concomitant injury in one-third of 39 patients.

c. *The extent of the lesion*

In the series of Parmley et al. (1958) the extent of the lesion in aortic rupture did not appear to have a significant bearing on survival. Nine of the 38 patients who survived the immediate effects of the trauma had complete transection of the aorta. Eight of the 9 survived longer than five days. However, in Strassmann's series (1947), the opposite seems evident: in 8 instances in which the victim lived for at least three hours after trauma, the rupture was incomplete as the adventitia was intact.

d. *Early detection and operation*

Early detection and operation will be discussed under the heading prognosis, chapter VI.

'In the diagnosis of TAR it is imperative to maintain a high index of suspicion and a constant awareness of the likelihood of this lesion in victims of high speed deceleration injuries, whether or not there is external evidence of chest injury' (Kirsh et al., 1976).

II.6.1. *Clinical symptoms*

The overwhelming majority of patients with TAR sustained injuries in vehicular accidents either as car-drivers, as passengers or as pedestrians. As a consequence, symptoms and signs of injury to more than one organ (skeleton, central nervous system, thoracic and abdominal organs) are commonly present. These other organ injuries will be discussed in chapter IV. Many patients are in shock, resulting from the associated injuries, and brain damage with coma is often seen (Plume and De Weese, 1979). Symptoms of chest injury are often absent, one third of the patients in the series of Parmley et al.(1958) had no external evidence of chest trauma. Barcia and Livoni (1983) found no significant difference in the frequency of clinical findings in thoracic trauma patients with and without TAR.
Clinical signs indicating thoracic trauma and aortic rupture are:
1) chest pain; 2) dyspnea; 3) dysphagia; 4) acute coarctation syndrome; 5) upper extremity hypertension; 6) paraplegia, anuria; 7) systolic murmer; 8) other findings e.g. hoarseness, cough, back pain.

1. *Chest pain*
Most often chest pain is caused by concomitant thoracic wall injuries e.g. fracture of the ribs, the sternum or the vertebrae, making this a rather aspecific sign. Chest pain specifically related to aortic rupture is descibed as interscapular or retrosternal as a result of dissection (Pate et al., 1968; Symbas, 1972; Kirsh et al., 1976; Marsh and Sturm, 1976).

2. *Dyspnea*
Dyspnea is another rather aspecific symptom. Obviously it is often due to concomitant thoracic injuries such as pulmonary contusion, aspiration and rib fractures with or without pneumo- or hemothorax.Compression of the trachea by the hematoma with resulting dyspnea can be a specific symptom.

3. *Dysphagia*
Dysphagia may be the result of compression of the esophagus by a para-aortic hematoma (Parmley et al., 1958; McBurney and Vaughan, 1961; Symbas, 1972; Vasko et al., 1977; Barcia and Livoni, 1983). It is a rather uncommon finding in acute TAR, in chronic aneurysms this sign is more commonly present (Bennett and Cherry, 1967).

4. *Acute coarctation syndrome*

The clinical syndrome of acute coarctation was desribed first by Rice and Wittstruck (1951) and is reported subsequently by many other authors (Malm and Deterling, 1960; Spencer et al., 1961; Gwathamy and Byrd, 1964; Pate et al., 1968; Flaherty et al., 1969; Langlois et al., 1971; Symbas, 1972; Griffin et al., 1973; Gazzaniga et al., 1975; Motin et al., 1980; Fisher et al., 1981). The syndrome is defined as upper extremity hypertension combined with lower extremity hypotension.

Symbas (1972) reported a diagnostic triad, that in his opinion is typical for acute aortic rupture : 1) increased blood pressure and pulse amplitude in the upper extremities; 2) decreased blood pressure and pulse amplitude in the legs; 3) a widened mediastinum on the chest film.

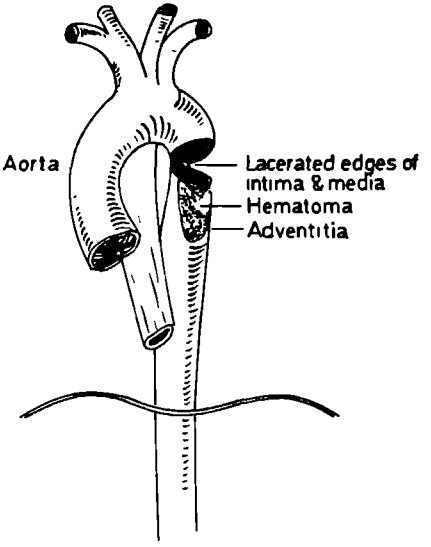


Figure II.3 Narrowing of the aortic lumen with resulting acute coarctation syndrome through subadventitial hematoma (Modified from Faraci and Westcott, 1977).

The ethiology of the syndrome is still a matter of controversy. According to Symbas (1972), the difference in blood pressure between the upper and lower extremities results from partial or total occlusion of the aorta by an intimal flap and/or by compression of the aortic lumen by an adjacent hematoma (Fig. II.3). Five of Symbas' seven cases showed this triad but his discription on the surgical findings failed to document this ethiology.

Keen (1972) proposed occlusion or a sympathic reflex inducing spasm of the aortic wall as an ethiologic factor, but observed no increased blood pressure in his own cases. A large variation in frequency of the acute coarctation syndrome exists in the reported series, see table II.6.

Table II.6 Frequency of acute coarctation syndrome in TAR

Year	*Author*	*Frequency*	
1969	Thomford et al.	2/7	23%
1971	Langlois et al.	2/6	33%
1972	O'Sullivan et al.	2/12	17%
1972	Symbas*	34/105	32%
1974	Fleming and Green	5/10	50%
1975	Appelbaum et al.	1/8	6%
1976	Kirsh et al.	16/43	37%
1977	Vasko et al.	7/9	78%
1980	Motin et al.	24/36	67%
1981	Fisher et al.	2/47	4%

* Collective review

5. *Paraplegia, anuria*

Paraplegia and anuria resulting from hypoperfusion and ischemia distal to the site of the rupture are less commonly encountered sequelae of this injury, see table II.7.

Table II.7 Frequency of paraplegia/anuria in TAR

Year	*Author*	*Paraplegia Frequency*		*Anuria Frequency*	
1961	Spencer et al.	1/6	16%		
1965	DeMuth et al.	1/1			
1968	Herendeen and King	1/1			
1968	Pate et al.	2/7	29%	2/7	29%
1971	Langlois et al.	1/6	16%		
1972	Symbas*	10/105	10%		
1976	Kirsh et al.	2/43	5%	1/43	2%
1977	Vasko et al.	1/19	5%	1/19	5%
1980	Motin et al.	0/36	0%	0/36	0%
1982	Barcia and Livoni	3/23	13%		

* Collective review

The anterior spinal artery in the thoracic region of the spinal cord usually receives two tributaries of inconstant origin. These tributaries are derived from spinal branches of the intercostal arteries of Th7-Th9. If these vessels are blocked ischemia of the spinal cord occurs (Brewer et al., 1972).

Compression of intercostal arteries by mural and mediastinal hematoma with subsequent ischemia of the spinal cord has been described as cause of the paraplegia

(Hughes, 1964). Also infolding of intima and/or media with subsequent (partial) occlusion of the aortic lumen is described as an ethiologic factor both for anuria and paraplegia (Symbas, 1972).
The presence or absence of paraplegia should always be verified before surgery by careful neurologic examination, because paraplegia can occur after surgery.

6. *Upper extremity hypertension*
Upper extremity hypertension is sometimes present as a rather specific clinical finding in aortic rupture, especially if it is coupled with evidence of blood loss (Jahnke et al., 1964; Laforet, 1965). It occurred in 43% of patients reported by Kirsh et al.(1976) and in 72% of 31 patients reported by Turney et al. (1976).
Motin et al. (1980) reported in 6 of 36 patients (15%) hypertension. This hypertension may be secondary to stretching or stimulation of the cardiac plexus that is located in the area of the isthmus (Lioy et al., 1974; Fox et al., 1979), or it may be caused by the same mechanism that produces the acute coarctation syndrome.

7. *Systolic murmur*
A systolic murmur over the precordium or subscapular area may be heard in patients with TAR. It is caused by disturbed patterns of blood flow in the traumatised segment of the aorta (Mc Clenathan and Brettschneider, 1965; Rittenhouse et al., 1969; Langlois et al., 1971; O'Sullivan et al., 1972; Symbas, 1972; Kirsh et al., 1976). Langlois et al.(1971) described this murmur as of low intensity and short duration. It is not an infrequent finding: an incidence is reported of 12-60% (O'Sullivan et al., 1972; Symbas, 1972; Kirsh et al., 1976; Motin et al., 1980; Barcia and Livoni, 1983).

8. *Other less frequent clinical findings*
Hoarseness and cough occur more frequently with chronic aortic aneurysms than in acute cases (Bahnson, 1953; McBurney and Vaughan, 1961). It is caused by encroachment of the left recurrent laryngeal nerve or left main stem bronchus by hematoma (Clarke, 1964). Sometimes it can be present as a symptom of acute rupture (Schwartz et al., 1975; Vasko et al., 1977).
Transient superior vena cava obstruction can occur especially when the ascending aorta is involved (Stoney et al., 1959; Wilson and Turnbridge, 1972). Another clinical finding sometimes associated with rupture of the ascending aorta is *acute aortic insufficiency* (Mc Clenathan and Brettschneider, 1965).
Signs and symptoms of *cardiac tamponade* may occur secondary to rupture of the intrapericardial portion of the ascending aorta (Pickard et al., 1977).
Retropleural dissection of hematoma is probably the cause of *back pain.* It is analogous to the retroperitoneal leakage of blood which causes such intense back pain in rupturing abdominal aneurysms (Eiseman and Rainer, 1958; Wilder, 1964; Lewis, 1965; Keen, 1972; Vasko et al., 1977).

Unfortunately, in most patients there are no physical signs or symptoms that point to the underlying vascular injury; therefore the diagnosis of rupture of the thoracic aorta depends largely on the recognition of mediastinal hemorrhage from the initial chest radiograph and confirmation by thoracic aortography (Woodring and Dillon, 1984).

II 6.2. *Chest radiography*

Introduction

More important than the clinical findings and a history of a deceleration trauma, is the role of the chest roentgenogram in suggesting a diagnosis of TAR. High KV technique (125 KV) and grid have to be preferred for the chest X-ray (Heystraten, 1980). The literature describes numerous plain chest roentgenographic findings in patients with TAR; these findings are the result of changes in the contours of the mediastinum by the local or more generalised hematoma resulting from rupture of the aorta and/or para-aortic mediastinal tissues and vessels.

As a hematoma caused by rupture of venous or small arterial vessels will also alter the mediastinal contours, the specificity of this chest X-ray finding for TAR is described as rather low. We will discuss the most frequent findings on the chest roentgenogram of patients with TAR separately: 1) mediastinal widening; 2) mediastinal chest width ratio; 3) displacement of the trachea to the right; 4) displacement of the left main stem bronchus; 5) displacement of the nasogastric tube; 6) loss of sharpness of aortic contours; 7) broadening of the right paratracheal stripe; 8) displacement of the right paraspinal line; 9) displacement of the left paraspinal line; 10) fractures of the first or second rib; 11) other rib fractures; 12) extrapleural apical cap and 13) other findings.

1. *Mediastinal widening*

In the past emphasis has been placed on mediastinal widening as the most important radiological finding on the chest roentgenogram in patients with aortic rupture (Freed et al., 1968; Kirsh et al., 1970; 1976; Rhys-Davies and Roylance, 1970; Attar et al., 1972; Lacquet et al., 1972; 1973; 1978; Wilson et al., 1972). Mediastinal widening in TAR is the result of blood accumulation in the mediastinum evoked by (partial) aortic wall rupture or damage to periaortic tissues or vessels (fig. II.4). Determining whether the mediastinum is truly widened is the most difficult aspect in evaluating chest X-rays of severely injured patients. The reason is that the initial chest film is generally made in a supine position, with short (100-110 cm) film-focus distance, with suboptimal inspiration and/or asymmetric positioning of the patient. These factors may cause a widening of the mediastinal shadow. Ayella et al.(1977) advocate the erect chest radiograph in trauma patients; however this will often be impossible. When possible, it should be done, because it makes evaluation of the

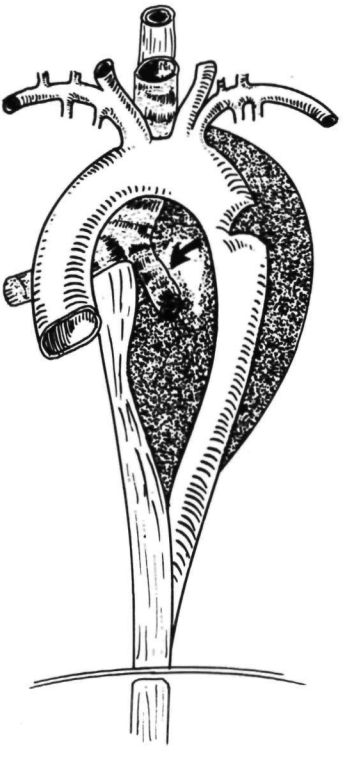


Figure II.4 Changes of mediastinal contours by mediastinal hemorrhage caused by (partial) aortic wall rupture or damaged periaortic tissues. Displacment of the esophagus and trachea can follow (Modified from Hermanutz and Bücheler, 1974).

mediastinum more accurate. Another problem in the evaluation of the widened mediastinum is the definition of (ab)normal mediastinal width. Marsh and Sturm (1976) stated that the mean mediastinal width on 100 cm supine AP chest films was 6.1 cm (range 3.5-8.5) for patients without chest trauma, 7.8 cm (range 6-10 cm) for patients with chest trauma and normal aortograms and 9.7 cm (8-12 cm) for patients with ruptured aortas. Barcia and Livoni (1983) found a mean mediastinal width of 9.8 cm (8.0-13.2)* in patients with aortic rupture versus 8.0 cm (5.2-13.5) in trauma patients with negative angiograms. The range of values for mediastinal width in the different groups overlap sufficiently to make mediastinal widening a nonspecific sign of aortic rupture; however, Marsh and Sturm (1976) consider a width of the mediastinum greater than 8 cm as an absolute indication for angiography. ln patients with TAR, a normal mediastinal contour is sometimes present because rupture can occur without mediastinal bleeding of any significance (Flaherty et al., 1969; DeMeules, 1971; Appelbaum et al., 1976; Ayella et al., 1977). On

* Measured at the level of the aortic knob on 100 cm supine AP chest radiographs.

occasion no mediastinal widening or other signs indicating aortic rupture are seen, therefore some authors (DeMeules, 1971; Wilson and Turnbridge, 1972; Griffin et al., 1973) feel that the indication for performing aortography for suspected aortic injury should include such findings as fractured first rib, sternal fracture, scapular fracture, severely displaced fractured clavicle or flail chest, since these injuries are often seen in association with aortic transection. However, Shackford et al. (1978) in a series of 14 patients with severe chest wall injury without widening of the mediastinum or other suggestive radiographic abnormality, did not diagnose a single aortic arch injury. They feel that severe chest injury is not an indication for aortography, unless associated with a widened mediastinum or specific physical findings indicating aortic trauma.

Sometimes, although infrequently, bleeding occurs later and an abnormal mediastinal contour is seen on serial chest films (Fleming and Green, 1974; Akins et al., 1981).

On the other hand, as previously mentioned, mediastinal widening can occur without injury of the aorta due to mediastinal bleeding from small superior mediastinal veins or arterial branches. Bleeding from the mammarian or intercostal arteries (or branches) or from the marrow of a fractured sternum is another possibility (Sandor et al., 1967; Kirsh et al., 1970; Attar et al., 1972; Tisnado et al., 1977). In a review of 16 patients with clinically and radiographically diagnosed mediastinal hemorrhage, Sandor et al. (1967) found that the bleeding was secondary to aortic rupture in 2 patients (12.5%) and was non-aortic in 14 patients (87.5%).

Since the plain chest radiograph cannot distinguish patients with mediastinal hemorrhage secondary to aortic rupture from those with non-aortic causes of mediastinal hemorrhage, aortography should be performed unless the mediastinum is unequivocally normal (Fisher et al., 1981).

A large number of negative aortograms should be accepted as standard practice (Woodring and Dillon, 1984).

In spite of the above mentioned problems in the evaluation of the mediastinal shadow on chest films of trauma patients, the widened mediastinum is an important sign (Fishbone et al., 1973; Vasko et al., 1977). The widening may be unilateral or bilateral, may have straight, smooth, sharply defined borders or may be irregular with a suggestion of lobulation. Frequently the contour of the aortic knob cannot be distinguished in the mediastinal shadow.

Few authors give absolute figures in the evaluation of the mediastinum in TAR. The series of Fisher et al. (1981) is one of the largest reported in the literature ($n = 47$). A mediastinum widened more than 8 cm was found in 75.5% of their patients with TAR, in 24.5% it measured less than 8 cm. In Barcia and Livoni's (1983) report all of the 14 patients with TAR had a mediastinal widening of more than 8 cm. This sign was one of the few chest film findings that was significantly more frequent in patients with TAR than in patients without TAR ($p < 0.05$). Many other authors (Kirsh et al., 1970; 1976; Symbas, 1972; Fleming and Green, 1974; Turney et al.,

1976; Tisnado et al., 1977; Motin et al., 1980; Peters and Gamsu, 1980; Gundry et al., 1983) found in all their patients a widened mediastinum, although they stated no absolute figures. Flaherty et al.(1969) described a normal mediastinal shadow in one of their eight patients as did Akins et al. (1981) in 2 of 44 patients with TAR. These patients developed a widened mediastinum after some days. Sturm et al. (1979) found a mediastinum wider than 8 cm in 11 of 18 patients (mean 8.8 cm). Schumacher et al. (1983) found in a review of the literature a widened mediastinum in 68% of cases with TAR and in their own series of 11 patients in 91%.

DeMeules et al. (1971) stated that other criteria for angiography than a widened mediastinum have to be developed because only 6 of their 12 patients with TAR showed a widened mediastinum. 5% of Akins et al. (1981)'s patients had a normal mediastinum on admission and developed signs of aortic trauma 6 to 36 hours later, indicating that all patients with severe chest trauma must have multiple chest X-rays during the first 48 hours after injury (Kingma 1981).

Data concerning the presence of a widened mediastinum in trauma patients without TAR are found in the papers of Kirsh et al., 1970; Tisnado et al., 1977; Fisher et al., 1951; and Barcia and Livoni (1983). Figures vary between 30 and 100%.

Reviewing the literature, a widened mediastinum (8 cm or more) is frequently present in trauma patients with TAR, but because an erect chest film generally cannot be obtained, technical factors can result in a 'widened' mediastinum. Evaluation of the mediastinal shadow is not always easy, especially in older patients, in whom unfolding of the aorta is another cause of widening the mediastinum. Mediastinal bleeding and subsequent widening can occur without TAR. Because of these factors, sensitivity of a widened mediastinum for TAR is high, specificity rather low.

2. *Mediastinal chest width (M/C ratio)*

In an effort to improve the specificity of mediastinal widening as a sign of TAR, Seltzer et al. (1981) made measurements of the mediastinal width (M) compared with the chest width (C) at three levels of the mediastinum. These measurements were made and ratios calculated at three levels: the aortic arch (level I), the mid-ascending aorta (level II) and the mid-descending thoracic aorta (level III). By using ratios, the effect of magnification is corrected. They compared the findings on 20 plain chest films of patients with TAR with findings on films of 20 trauma patients without TAR and with findings on 20 films of a control group. If an M/C ratio of 0.20 or above at level I is defined as abnormal, sensitivity for aortic rupture was 100%, specificity 15%. Increasing the treshold to an M/C ratio of 0.25 decreased sensitivity to 95%, while specificity was raised to 75%. If the treshold was 0.28, sensitivity was 85%, specificity 100%. Sefczek et al.'s (1983) report supports the superiority of M/C ratios over other advocated methods in determining mediastinal widening. Their study detected abnormal widening of the mediastinum in only 7% of the negative cases and in 90% of cases with an aortic rupture, while the M/C ratio was greater than 0.25 at the level of the aortic knob. Marnocha et al.(1984) were

unable to confirm the data of Seltzer et al. (1981). They found in 54 patients much higher M/C ratios at the level of the aortic knob: 0.39 in patients with TAR (n = 10) versus 0.36 in control patients (n = 44). On basis of their experience they found the M/C ratio unreliable in confirming or excluding aortic rupture.

3. *Displacement of the trachea to the right*

Conflicting data are found in the literature regarding the significance of displacement of the trachea to the right as a sign of aortic rupture (Fig. II.4). This can be explained partly by the findings at operation or autopsy: in some cases of aortic rupture a local peri-aortic hematoma at the isthmus displaces the trachea, while a more generalised mediastinal hematoma does not need to displace the trachea. If there is no significant hematoma, no displacement will occur. In evaluating tracheal deviation, one must exclude patient's rotation, right upper lobe collapse, a left sided tension pneumothorax, ectasia of the aortic arch, or some other thoracic process that may account for such deviation (Barcia and Livoni, 1983). Table II.8 summarizes data from the literature. As can be seen from table II.8 tracheal deviation to the right is present with great variation in the different series. It is by no means specific and in many cases it is absent as a sign of TAR. If it is present, however, it is an important sign for the further evaluation of these patients.

Table 11.8 Frequency of deviation of the trachea in patients with and without aortic rupture

Year	*Author*	*Frequency with TAR*		*Frequency without TAR*	
1967	Sandor	2/7	29%		
1969	Flaherty et al.	8/8	100%		
1972	Symbas	11/105	10%		
1973	Redman	5/8	63%	4/16	25%
1976	Dart and Blaitman	0/4	0%		
1976	Kirsh et al.	14/43	33%	0/13	0%
1976	Marsh and Sturm	5/5	100%	8/40	20%
1977	Tisnado et al.	7/7	100%	0/24	0%
1979	Sturm et al.	9/18	50%		
1980	Andresen	5/10	50%		
1980	Gerlock et al.	5/8	63%	2/38	5%
1980	Peters and Gamsu	6/14	43%	4/14	29%
1981	Fisher et al.	30/49	61%	6/19	31%
1983	Barcia and Livoni	2/17	12%	4/86	5%
1983	Schumacher et al*.	18/294	6%		
1984	Woodring et al.	8/15	53%	8/20	40%

* Collective review.

4. *Displacement of the left main stem bronchus*

Another indication on plain chest films of patients with TAR described in the literature is the downward displacement of the left main bronchus by the local periaortic hematoma or by the false aneurysm (Fig. II.4). In the most reported series this is evaluated as a subjective sign and no absolute measurements are given except by Marsh and Sturm (1976). They calculated left main bronchus angles from supine 100 cm AP chest films in patients with TAR, in trauma patients without TAR and in patients without trauma. Although they found no significant differences, all their patients with TAR had left main bronchus angles that were less than 40 degrees. Only 5 out of 100 patients without trauma had this finding, and they all had emphysema. Table II.9 gives the data from the literature.

From this table, it is evident that depression of the left main bronchus is less frequent than deviation of the trachea, but when it is present together with other signs, it is a valuable indicator of a localised mediastinal hematoma. Sensitivity and specificity of the sign for TAR by itself are low.

Table II.9 Downward displacement of left main bronchus in trauma patients with and without TAR

Year	*Author*	*Frequency with TAR*		*Frequency without TAR*	
1969	Flaherty et al.	2/8	25%		
1973	Redman	6/8	75%	4/13	30%
1976	Kirsh et al.	13/43	30%	4/13	30%
1976	Marsh and Sturm	4/5	80%	8/40	20%
1977	Tisnado et al.	2/7	28%	0/24	0%
1979	Sturm et al.	6/8	75%		
1980	Gerlock et al.	5/8	62%	2/38	5%
1980	Motin et al.	17/36	47%		
1981	Fisher et al.	26/49	53%	5/19	26%
1983	Barcia and Livoni	1/15	6%	0/86	0%
1983	Schumacher et al*.	9/294	3%		
1984	Woodring et al.	6/15	40%	2/20	10%

* Collective review

5. *Displacement of the nasogastric tube*

The esophagus closely follows the aorta at the level of the aortic arch and isthmus. A mediastinal hematoma can shift the esophagus to the right in case of an aortic rupture (Fig. II.6). This esophageal displacement can be demonstrated on an AP chest roentgenogram with an opaque nasogastric tube in the esophagus. The tube is displaced if it extends to the right of the spinous process at the level of the 4th thoracic vertebra. When both the nasogastric tube and the trachea are deviated to the right, Gerlock et al. (1980) give a 96% probability of aortic rupture. The sign

Table II.10 Nasogastric tube displacement in patients with and without TAR

Year	*Author*	*Frequency with TAR*		*Frequency without TAR*	
1977	McIlduff et al.	3/3	100%	0/34	0%
1977	Tisnado et al.	7/7	100%	0/24	0%
1980	Gerlock et al.	6/8	80%	0/38	0%
1981	Cole et al.	10/24	41%	9/50	18%
1981	Fisher et al.	18/27	66%	3/13	23%
1983	Barcia and Livoni	6/12	50%	5/52	9%
1983	Sefczek et al.	4/5	80%	–	–
1984	Woodring et al.	5/7	71%	2/12	17%

was first described by McIlduff et al. (1977) and Tisnado et al. (1977). Table II.10 gives figures from the literature.

Radiological findings thus indicate a sensitivity of about 60-70% in nasogastric tube deviation to the right for TAR, rather than the 80-100% suggested by the studies of Tisnado et al. (1977) and Gerlock et al. (1980). Nasogastric tube deviation also may be produced by patient rotation, left sided tension pneumothorax, right lung collapse and placement of the nasogastric tube so that its tip is pressed against the wall of the stomach. In order to evaluate nasogastric tube deviation, the tube tip should be positioned freely in the stomach (Gerlock et al., 1980). In the appropriate setting, nasogastric tube deviation is a moderately sensitive indicator of aortic rupture and indicates the need for angiography. However, absence of nasogastric tube deviation in no way excludes aortic rupture and the decision to perform angiography must be made on the basis of clinical and other radiographic findings.

Table II.11 Unsharp aortic contour in patients with and without TAR

Year	*Author*	*Frequency with TAR*		*Frequency without TAR*	
1969	Flaherty et al.	6/8	80%		
1973	Redman	7/7	100%	13/13	100%
1976	Kirsh et al.	41/43	95%	9/13	69%
1976	Marsh and Sturm	18/18	100%		
1979	Sturm et al.	4/5	80%		
1980	Gerlock et al.	6/8	80%	15/38	39%
1980	Motin et al.	32/36	88%		
1982	Fisher et al.	37/49	76%	14/19	74%
1983	Barcia and Livoni	17/17	100%	68/86	79%
1983	Schumacher et al.	11/11	100%		
1983	Sefczek et al.	10/10	100%		
1984	Woodring et al.	8/15	53%	9/20	45%

6. *Loss of sharpness of aortic contours*

This is a rather aspecific finding in cases of TAR as it reflects any mediastinal hematoma or fluid collection. Its frequency is high in patients with TAR; however,it is also often present too in patients without aortic rupture.Table II.11 provides data from the literature.

From table II.11 can be concluded that an abnormal aortic outline is a nonspecific finding on plain chest films of patients with TAR. On the other hand, a clear aortic outline virtually excludes the possibility of an aortic tear of the isthmus (Barcia and Livoni, 1983). The aortic outline may also be indistinct if the radiograph is underexposed or if there is pleural effusion, pulmonary contusion or collapse.

7. *Broadening of the right paratracheal stripe*

The right paratracheal stripe is produced by the interface between the right lung and the right lateral wall of the trachea in the supraazygos recess (Heitzman, 1977). The right paratracheal stripe is composed of three tissue planes: a) the pleura and pleural space; b) the mediastinal connective tissue and its contents; c) the tracheal wall. This stripe is visible in 82-94% of normal patients and normally measures at a level 2 cm above the azygos arch less than 5 mm on frontal radiographs (Savoca et al., 1977). Widening of the paratracheal stripe to 5 mm or more was found by Woodring et al. (1982) to be a reliable indicator of abnormality, including mediastinal hemorrhage. They found no cases of arterial injury in 29 patients with a normal paratracheal stripe of less than 5 mm. In 48 patients with an abnormal stripe of more than 5 mm, they found 11 cases (23%) of arterial injuries (8 aortic ruptures, 3 brachiocephalic ruptures). They conclude that widening of the stripe is a strong indicator of vascular injury in the mediastinum.

8/9. *Displacement of paraspinal lines*

It has been suggested by Fraser and Paré (1978) that the right paraspinous interface is normally not visible on plain chest radiographs except when large osteophytes are present. This is confirmed by Peters and Gamsu (1980). The anatomy of the right paraspinous interface is best understood by comparing this area on the chest radiograph with its appearance on an axial coupe of a normal chest (Fig. II.5). On the right, the posterior visceral and parietal pleura are in close contact with the right margin of the vertebral body and pass forward in front to become the azygoesophageal interface. Because of the oblique orientation , these interfaces are less conductive to imaging. On the left, the pleura is not in contact with the margin of the vertebral body, but passes anteriorly and laterally around the aorta. Because they are oriented in a plane parallel to the X-ray beam in the AP projection, they are optimally imaged (Heitzman, 1977). Genereux (1983) held the opinion that paraspinal lines are mach bands, which can be defined as edge-enhancing phenomena created by the retina in response to strong differences in transmitted illumination.

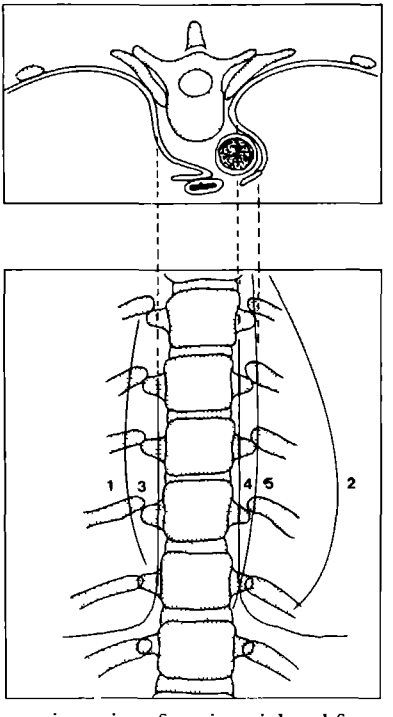


Figure II.5 Anatomy of paraspinous interfaces in axial and frontal projection. Dotted lines indicate anatomical substrates of pleural lines and aortic lines in cross section (Modified from Lachman, 1942). (1) and (2), cardiac contours, (3) right paraspinal line,(4) left paraspinal line,(5) aortic contour.

The position of the normal left paraspinous interface varies, being more lateral in some patients with elongated aortas. This variability can make assessment of a possible displacement difficult.

Peters and Gamsu (1980) considered the paraspinous interfaces displaced if they were separated from their normal close relationship with the lateral margin of the adjacent vertebral bodies. This can be caused by posterior mediastinal hematoma. (Displacement can also be seen with hematomas originating in the spine, but bone changes often provides clues indicating the diagnosis.)

The left paraspinous interface is considered displaced if it is located at a distance greater than one half of that from the spine to the left margin of the descending aorta (Peters and Gamsu, 1980).

For data regarding displacement of paraspinal lines in patients with TAR, see table II.12.

Displacement of paraspinal lines is another finding on the plain chest films of trauma patients with rather low specificity and sensitivity which merely indicates a mediastinal hematoma; however it is regarded sufficient by itself as an indication for angiography.

Table II.12 Displacement of paraspinal lines in patients with and without TAR

Year	*Author*	*Frequency with TAR*			*Frequency without TAR*		
1973	Redman	Le	7/8	88%	Le	4/16	25%
1976	Marsh and Sturm	Ri	0/5	0%	Ri	0/5	0%
1979	Sturm et al.	Ri	3/18	16%			
		Le	3/18	16%			
1981	Fisher et al.	Ri	5/49	10%	Ri	0/14	0%
		Le	6/49	12%	Le	3/12	25%
1982	Peters and Gamsu	Ri	8/14	57%	Ri	0/14	0%
		Le	5/9	55%	Le	3/14	21%
1983	Barcia and Livoni	Ri	4/14	28%	Ri	12/85	14%
		Le	10/12	83%	Le	19/79	24%
1984	Woodring et al*.		6/15	40%		5/20	25%

* Not defined right or left

10. *Fractures of the first or second rib*

Fractures of the first or second rib after blunt thoracic trauma indicate a severe injury (Richardson et al., 1975). Patients who have sustained a fracture of the first ribs have an 8-14% incidence of associated vascular injury, either of the aorta or of the brachio-cephalic vessels (Thomas et al., 1978; Philips et al., 1981; Livoni and Barcia, 1982; Woodring et al., 1982). Some authors have stated that fracture of the first rib alone is an indication for angiography (DeMeules et al., 1971; Richardson et al., 1975). Other, more recent reports (Woodring et al., 1982; Gundry et al., 1983; Kirshner et al., 1983) found isolated first and second rib fractures not to be an indication for thoracic angiography. In a group of 105 trauma patients reported by Woodring et al. (1982), angiography revealed a major arterial injury in 8 out of 55 patients (14%) without first or second rib fracture compared with 4 out of 50 (8%) in those patients with first or second rib fracture. They stated: 'since a higher percentage of major arterial injury in our series was present in those patients without upper rib fractures than in those patients with fractures, we believe that first and second rib fractures have no predictive value for arterial injury'. Kirshner et al. (1983) reported that the probability of sustaining TAR is the same for patients with upper rib fracture as for those with other rib fractures. Their data also failed to show a statistical difference in the incidence of TAR associated with upper rib fractures when compared to absence of other rib fractures.

Gundry et al. (1983) reported a low diagnostic accuracy and no statistical correlation between first or second rib fractures and TAR in 173 patients with blunt thoracic injury.

Table II.13 provides some data from recent reported series.

Table II.13 First or second rib fractures in patients with and without TAR

Year	*Author*	*Frequency with TAR*		*Frequency without TAR*	
1981	Seltzer et al.	1/6	17%	63/207	30%
1982	Livoni and Barcia	2/22	9%		
1982	Woodring et al.	4/50	8%		
1983	Barcia and Livoni	7/17	41%	44/86	51%
1983	Gundry et al.	8/25	32%		
1983	Kirshner et al.	1/21	4%		

These figures show a rather great variability in the correlation of first and second rib fractures and TAR. In most patients there was a combination with signs of mediastinal hematoma and this combination especially makes angiography mandatory (Woodring and Dillon, 1984).

11. *Other rib fractures*

Conn et al.(1963) reported an 84% incidence of rib fractures in blunt thoracic trauma, which is so common that it is quite unlikely that a correlation with TAR can be made. Howell and colleages (1963), in a review of 100 patients with multiple rib fractures and flail chest, found no case of major thoracic arterial injury.Gundry et al.(1983) and Kirshner et al.(1983) reported far more multiple rib fractures in chest trauma patients who did not have TAR than in those who did, making this finding a poor predictor for aortic rupture.

12. *Extrapleural apical cap*

This sign on plain chest radiographs of patients with TAR was first described by Simone et al.(1975). At the isthmus of the aorta, where most of the ruptures occur, a potential space exists between the aorta and the parietal pleura of the left lung. If the parietal pleura is intact, blood may track cephalad along the course of the left

Table II.14 Apical cap in patients with and without TAR

Year	*Author*	*Frequency with TAR*		*Frequency without TAR*	
1975	Simone et al.	25/27	93%		
1977	Tisnado et al.	3/7	43%	9/24	38%
1980	Gerlock et al.	3/8	38%	24/38	63%
1980	Peters and Gamsu	4/9	45%	7/14	50%
1981	Fisher et al.	18/49	37%	8/19	42%
1983	Barcia and Livoni	10/16	63%	49/86	57%
1983	Gundry et al.	5/21	24%		
1984	Woodring et al.	8/15	53%	2/12	17%

subclavian artery between the parietal pleura and the extrapleural soft tissues, resulting in the radiographic finding of the extrapleural apical cap (Simone et al., 1975). Other causes for an apical cap are a local apical pleural thickening and hematoma from associated rib or clavicular fractures or bleeding following subclavian line insertion in the subclavian vein. The left apical cap can also be a normal shadow of the subclavian artery (Gondos, 1982).
The frequency of apical cap in TAR is shown in table II.14.
When present, the apical cap can be one of the earliest indicators of TAR. However, the left apical cap was never the only visible abnormality on the initial chest radiograph in positive patients (Simone et al., 1975). Barcia and Livoni (1983) found no significant difference in frequency of occurence of this sign in patients with and without TAR.

13. *Other findings*
Some other infrequently present signs have been reported in the literature:
- displaced superior vena cava (Molnar and Pace, 1966; Marsh and Sturm, 1976; Sturm et al., 1979).
- opacification of the normal 'clear space' between the aortic knob and the left pulmonary artery (Marsh and Sturm, 1976; Sturm et al., 1979). No clear cut parameters have been put forth to help determine whether these signs are really present. Barcia and Livoni's data indicated that an opacified 'pulmonary window' occurred more frequently in patients with TAR compared with those without aortic injury (80% versus 45%). Displacement of the superior vena cava was not shown to be of significance.
  An unique sign in TAR, displacement of intimal calcification by subadventitial hematoma, was described by Gray and Kirsh (1975). Normally the distance between intimal calcification and adventitia is only a (few) millimeter(s). This sign is rather infrequently present because most aortic ruptures occur in young patients without calcifications.
- Anterior displacement of the trachea on the lateral chest radiograph was described by Molnar and Pace (1966). This is caused by a localised mediastinal hematoma. Because lateral chest radiographs are only infrequently obtained in trauma patients, this sign is not often seen.

II.6.3. *Angiography*

Only angiography can establish a definitive diagnosis of aortic rupture. Most authors agree that angiography is indicated by priority when TAR is suspected. There are many reasons for this. The most important is the low specificity of clinical and plain film findings in TAR. Exact localisation of the rupture is important for the surgeon: tears may be multiple (Greendyke, 1966; Chimochowski et al., 1973;

Fisher et al., 1981) or there may be associated lesions of the brachiocephalic vessels which may dictate another operative approach (Rhys-Davies and Roylance, 1970; Fishbone et al., 1973); further, sometimes anatomical variations such as an arteria lusoria may be present (Rosenbusch et al., 1976).
According to Bodily et al. (1977), TAR was confirmed by angiography in approximately 15% of the patients who underwent angiography for suspected aortic rupture. Richardson et al.(1979) performed 167 angiographies in trauma patients to investigate the possibility of TAR, in 29 (17%) TAR was demonstrated.

II.6.3.1. *Technique of angiography*

The first reported angiography in a patient with a traumatic aortic aneurysm was performed by an intravenous contrast injection (Steinberg, 1957). Subsequent authors have used several alternative methods including transventricular (Wilder, 1964), transseptal (Jahnke et al., 1964), brachial and femoral puncture (Blazek, 1965). Injection in the arterial tree is considered more desirable than a venous approach. In the earlier literature (Molnar and Pace, 1966; Freed et al., 1968; Lipchik and Robinson, 1968; Flaherty et al , 1969; Hermanutz and Bucheler, 1972), the preferred method was the right axillary or brachial approach due to the fear for traversing the area of aortic injury; in later reports (Blazek, 1965; Fishbone et al., 1973; Rhys-Davies and Roylance, 1970; Kirsh et al., 1976; Ayella et al., 1977; Fisher et al., 1981) the femoral route is preferred.
The femoral approach compared with the axillar or brachial one, has several advantages; it is familiar to most radiologists, it is quick, and more convenient for the patient, further the complication rate is lower (Abrams, 1983). The femoral approach is more flexible, as it allows angiographic evaluation of other areas or organs with suspected injury as the liver, spleen or kidneys. Most authors use a J guide wire and a pigtail catheter to cross the aortic tear. Thirty to forty ml. of contrast medium (300-360 mg I/ml) is injected for 2 seconds in the ascending aorta by a pressure device. A high rate of injection and recording is necessary to prevent excessive dilution of contrast medium as a result of increased cardiac output in severely injured patients.
The left anterior oblique position (60-70°) is optimal for visualisation of the aortic arch and the proximal part of the descending aorta. The proximal parts of the great vessels can be evaluated adequately in this projection (Eller and Ziter, 1970; Fisher et al., 1981).
The axillary or brachial approach is the preferred method when the femoral pulses are diminished or absent, since the aorta may be completely transected in such cases (Fishbone et al., 1973). In those cases it is often impossible to traverse the lesion from below.

The great majority of aortic ruptures in clinical patients are found in the aortic isthmus (Symbas, 1972). The radiographic findings on the aortogram are:

a. false aneurysm
b. radiolucent lines
c. aortic wall dissection

a. *False aneurysm*

An image of a false aneurysm is the result of subadventitial or subpleural extravasation of the contrast agent through the traumatic defect in the intima and media or all three layers of the aortic wall, producing a fusiform or saccular periaortic hematoma. The contrast medium usually remains within this false aneurysm after the remaining part of the aorta has been cleared of contrast medium. A sharply defined linear defect (intima and/or media) can sometimes be seen in the contrast column at the proximal or distal margin of the aneurysm. This defect is produced by the protusion of the transected portion of the aortic wall in the aneurysm. Frank extravasation may be present when the adventitia is defective (Freed et al., 1968; Fisher et al., 1981). Sometimes the aorta is almost completely occluded by an infolded torn wall or a subadventitial hematoma.

b. *Radiolucent lines*

These lines (partial or circumferential) represent intimal and/or medial flaps extending into the lumen of the opacified aorta and can be the only signs of aortic wall rupture.

c. *Aortic wall dissection*

Several authors (Samson, 1931; Rice and Wittstruck, 1951; Malm and Deterling, 1960; Griffin et al., 1973; Faraci and Westcott, 1977) have described subadventitial dissection caused by a subadventitial hematoma. This subadventitial hematoma can be the cause of an acute coarctation syndrome or the hematoma may rupture into the pleura (Malm and Deterling, 1960; Gwathmay and Byrd, 1964).

Usually the dissection extends over a rather short distance, in contrast with the classical dissection in which the hematoma is localised in the media and where longer segments of the aorta are involved.

Faraci and Westcott (1977) described three older patients with classical media dissection in whom in their opinion trauma was the causative factor. This seems to occur only in patients with pre-existent atherosclerosis. The only other case in which media dissection caused by trauma is reported, was in a pregnant woman (Rice and Wittstruck, 1951); however during pregnancy there is an increased incidence of spontaneous dissection (Anagnostopoulos et al., 1972). Normally the contours of the aorta are perfectly smooth. Awareness of the appearance of the ductus diverticu-

lum is of importance in the angiographic evaluation of the aorta. The ductus diverticulum is a focal bulge along the antero-medial aspect of the aortic isthmus at the site of the obliterated ductus arteriosus.
Abrams (1983) described a ductus diverticulum in as many as 33% of thoracic aortograms of children. In adults, Goodman et al. (1982) found a ductus diverticulum in 9% of 103 thoracic aortograms. In differentiating a ductus diverticulum from a posttraumatic pseudo-aneurysm, the most important feature is the lack of a radiolucent filling defect in the presence of a ductus diverticulum. Another differentiating feature is, that there is no delay in washout of the contrast medium in a ductus diverticulum which is often the case in posttraumatic pseudo-aneurysm.
A few other conditions that enter into the differential diagnosis are physiological streaming of contrast, localised dissecting aneurysm, and aneurysms secondary to atherosclerosis or syphilis.
It is often impossible on the basis of the angiographic study to differentiate between partial and complete circumferential laceration of the aorta (Freed et al., 1965). Figure II.6 a-f shows a normal image of the aortic arch and several examples of a ductus diverticulum.

d. *Localisation*
In clinical series most ruptures are found at the classical localisation, the isthmus. This is the region between the left subclavian artery and the ligamentum arteriosum. 90-95% of aortic ruptures are found in this area (DeMeules et al., 1971; O'Sullivan et al., 1972; Symbas, 1972; Appelbaum et al., 1975; Thevenet, 1975; Kirsh et al., 1976; Bodily et al., 1977; Turney et al., 1977; Vasko et al., 1977; Plume and de Weese, 1979; Richardson et al., 1979; Akins et al., 1981; Barcia and Livoni, 1983). Only Fisher et al. (1981) in an angiography series found a lower figure: 79% at the isthmus, 15% in the descending aorta between the ligament and the diaphragm.

In the literature some reports of cases of isolated ascending aortic rupture have been published (Beall et al., 1964; Freed et al., 1965; Matloff and Morton, 1965; Bross, 1971; Appelbaum et al., 1975; Charles et al., 1977; Akins et al., 1981). Symbas (1973) reported 105 clinical cases: 94 were localised at the isthmus, 3 in the ascending aorta, 3 in the descending thoracic aorta, 2 in the aortic arch. Ruptures of the descending thoracic aorta are relatively rare: Plume et al.(1972) found 1 case in 20 patients, Kirsh et al.(1976) one in 44 patients. Multiple ruptures are not infrequent: Turney et al.(1976) reported 7 patients with multiple ruptures in 31 cases; Bodily et al.(1977) reported 3 cases of multiple tears in 39 patients, all localised at the isthmus. In case of multiple ruptures various combinations can be present: Blazek et al., (1965); Matloff and Morton, (1968); Fishbone et al. (1973); Fleming and Green, (1974); Kirsh et al. (1976).
The most frequent localisation was distal from the ligamentum arteriosum in the ventral aortic wall (Strassmann, 1947; Gerbode et al., 1957; Parmley et al., 1958).

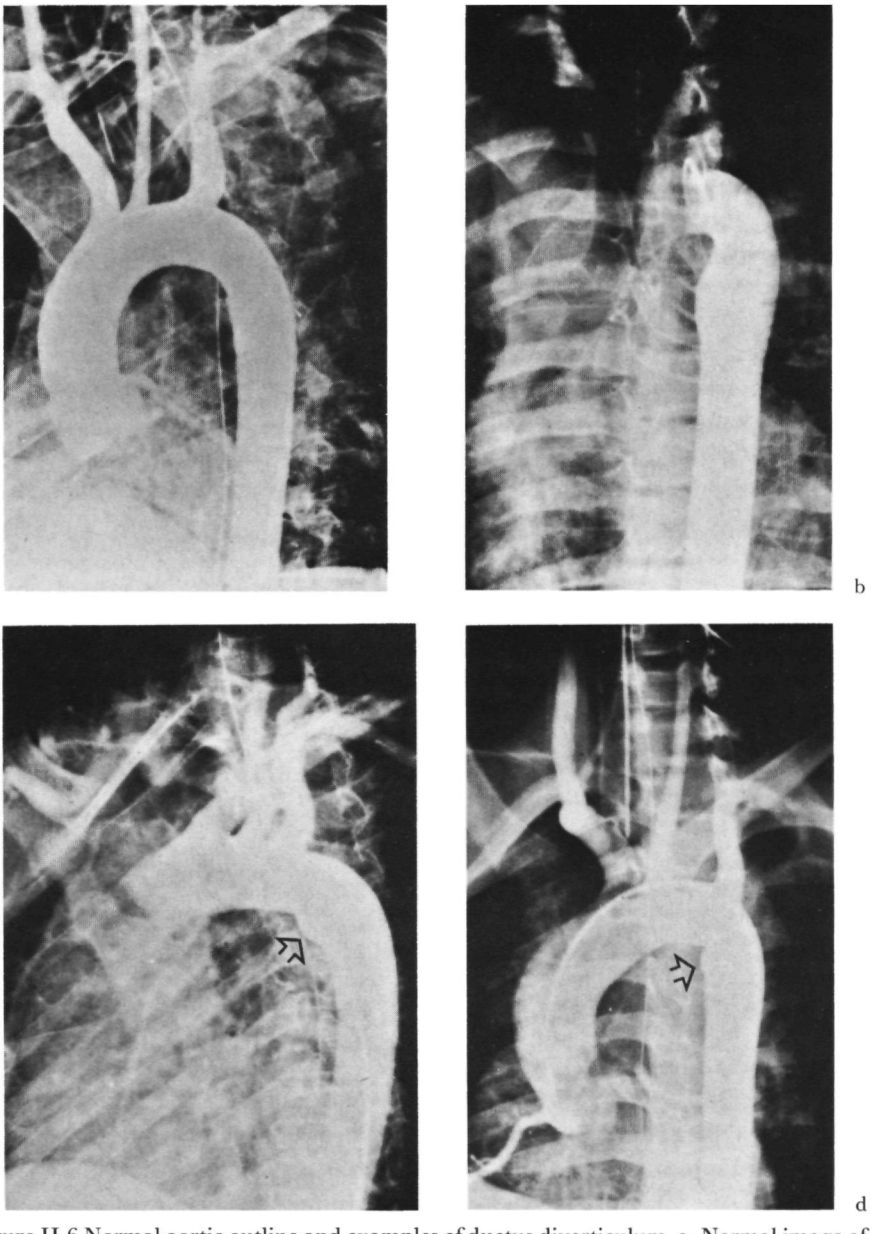
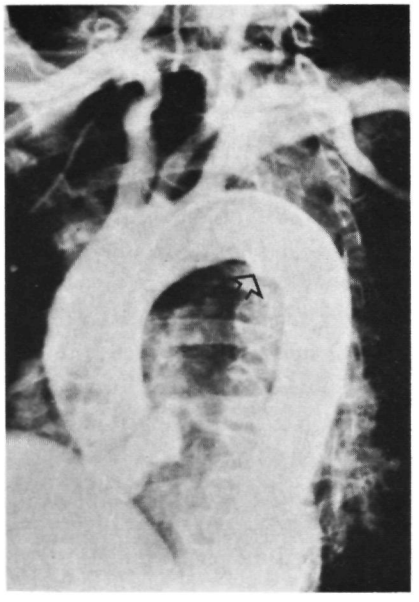


Figure II.6 Normal aortic outline and examples of ductus diverticulum. a. Normal image of the aortic arch and descending aorta: smooth, straight contours. b. Diffuse widening of the proximal descending aorta: normal variant (Contrast medium had already been flushed from ascending aorta). c.-e. Localised bulging of aortic contour at the level of the former ductus arterious (arrow). f. Older patient with elongated aorta: small contour irregularity at the site of the ductus arteriosus attachment (arrow). Note that in none of the images an intimal flap can be seen.



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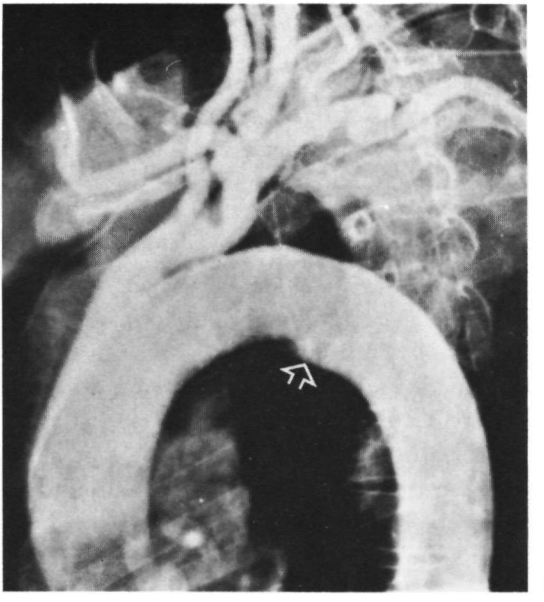


f

II.6.3.3. *Complications of angiography*

Generally the risks due to an angiographic investigation are the same in patients with and without TAR.

Although it is conceivable that a complete rupture of the aorta might occur while the patient is on the angiography table or immediately after the study, a cause and effect relationship between angiography and this terminal event has never been reported or suggested (Fisher et al., 1981). Until the present, no reports are known where catheter or guide wire manipulation has led to perforation of the aortic wall or aneurysm with subsequent exsanguination and death. Femoral arteriography carries a lower complication rate compared with brachial or axillary arteriography (Abrams, 1983), which should only be performed when the femoral pathway is not available for catheterisation because of disease, direct trauma to the groin or diminished femoral pulses as is the case in aortic transection (Fisher et al., 1981). Speed in achieving an accurate diagnosis is critical; prolonging the diagnostic study is far more hazardous to the patient than the theoretical hazard of the technique itself.

II.6.4 *Use of newer modalities (DSA, CT) in patients with aortic rupture*

1. *Computed tomography (CT)*

Although 3th and 4th generation CT scanning is available since 1978, only two reports on the use of CT scanning in TAR are published (Toombs et al., 1981;

Heiberg et al., 1983). The CT findings of an injured aorta were 1) false aneurysm; 2) linear lucency within the opacified aortic lumen caused by the torn edge of the aortic wall; 3) marginal irregularity of the opacified aortic lumen; 4) peri-aortic or intramural aortic hematoma; 5) dissection.

These scans were performed on clinical stable patients or when a CT study of the brain was the clinician's first priority.

Ten patients suspected to have an aortic tear were studied. Four had an aortic rupture diagnosed by CT, confirmed by angiography. Artifacts can be produced by external lines and monitoring leads; artifacts can also be produced when the patient is unable to raise his arms above the head or cannot hold his breath for 5 seconds. Contrast enhancement is essential in differentiating the aortic lumen from a surrounding mediastinal hematoma. Another drawback is that lesions of the brachiocephalic vessels cannot be detected.

Toombs et al. (1981) state: 'our experience thus far is inadequate to define the proper role of CT in suspected injuries to the great vessels: therefore aortography continous to be the preferred method in such cases'. Although CT scanning is capable of detecting aortic tears, the sensitivity of the technique, especially for smaller tears, is not yet known. It is not yet clear what the role of CT for diagnosing TAR in the future will be.

2. *Digital subtraction angiography (DSA, DVI)*

As a noninvasive angiographic method, DSA (DVI) was introduced a few years ago. By using digital transformation of a video signal and subtraction techniques, it is possible to make small contrast differences visible and to get an image of blood vessels by intravenous contrast injection. Principally, it is possible to obtain an adequate image of the aortic arch and brachio-cephalic vessels, however it is essential that there is no motion between the noncontrast image (used as a mask for the subtraction) and the contrast image. This will usually present a problem in many trauma patients, as breath holding is difficult or even impossible. In such cases DSA may not be useful. Intravenous or intra-arterial DSA can be an alternative method in patients who can cooperate well and can hold their breath. Rauber and Kollath (1983) reported 4 patients in whom a diagnosis of aortic rupture was made by DSA. The sensitivity in a larger group of patients has still to be evaluated.

OWN STUDY: CLINICAL AND ROENTGENOLOGIC FINDINGS OF 63 PATIENTS WITH ACUTE RUPTURE OF THE THORACIC AORTA AND COMPARISON OF THESE FINDINGS WITH 62 PATIENTS WITH BLUNT CHEST TRAUMA WITHOUT AORTIC RUPTURE

III.1. **Introduction**

In this chapter we will present our own experience with patients with acute traumatic rupture of the thoracic aorta (TAR). A retrospective analysis is performed on 41 patients referred to the University Hospital St. Radboud in Nijmegen and 22 patients referred to the University Hospital in Groningen between 1970 and 1984. We present and analyse the clinical and radiological data of these patients and compare the data with those of 62 patients with blunt chest trauma, but without a ruptured aorta.
We also compare our results with those presented in the recent literature.

III.2. **Own patients (Nijmegen and Groningen)**

The 63 patients under study comprise all successive patients with acute traumatic aortic rupture seen in the University Hospital in Nijmegen between 1970 and 1984 and between 1976 and 1984 in the University Hospital in Groningen. We reviewed the patient charts, chest X-rays and the angiograms of the thoracic aorta of these patients. In the same period we have seen 11 patients with a chronic traumatic aneurysm (these patients will be discussed in the chapters VII and VIII).

Table III.1 Patients with TAR

Nr. patients	*Nijmegen* 41*	*Groningen* 22	*Total* 63
Male	30	18	48
Female	11	4	15
Mean age (years)	36	38	36
Age (range)	17-76	17-66	17-76

* Including 6 patients with a chronic traumatic aneurysm, from whom the data of the initial trauma were available.

Table III.2 *Patients with blunt chest trauma without TAR*

	Nijmegen	*Groningen*	*Total*
Nr. patients	45	17	62
Male	34	14	48
Female	11	3	14
Mean age	39	41	39.7
Age (range)	15-83	13-83	13-83

When possible, the available data at the time of the initial trauma (n = 6) from this last group of patients were evaluated together with the acute group. Because of the retrospective character of the study, not all data are complete.
For comparison of the clinical data and the radiological figures of the patients with TAR, we reviewed the clinical records, chest radiographs and angiograms of 62 patients who underwent aortic arch angiography for hemomediastinum as a result of closed chest trauma, but with negative angiographic findings (these patients will figure as control group).

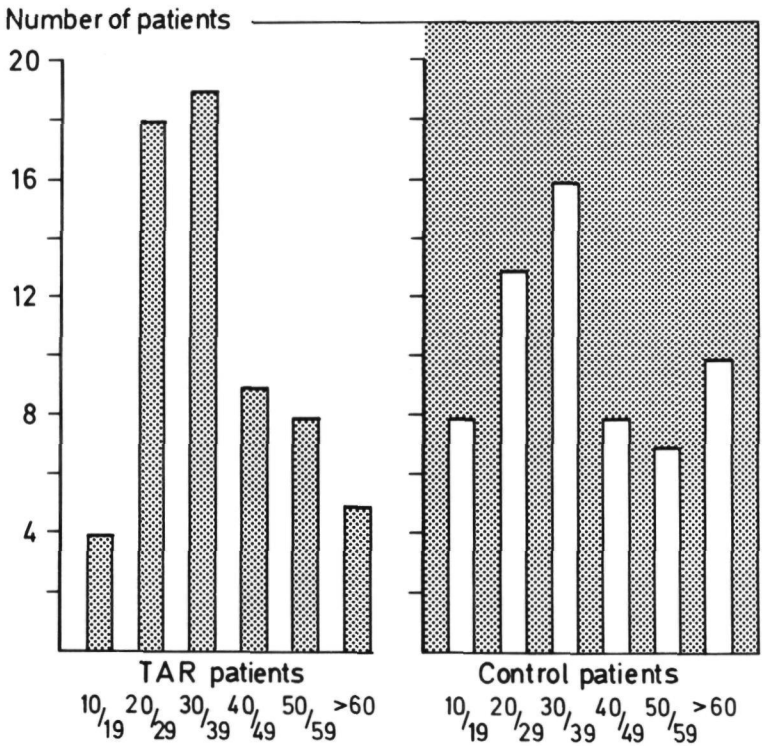


Figure III.1 Age distribution of TAR (n = 63) and control (n = 62) patients.

Table III.1 provides data regarding number, sex and (mean) age of the patients with TAR.
Table III.2 shows data regarding number, sex and (mean) age of the patients without TAR (the controls).
The groups are comparable as regard to sex and age distribution ($P > 0.10$).
In figure III.1 the age distribution of both groups is shown.
The type of trauma of both groups is shown in table III.3.

Table III.3 *Estimated main direction and type of trauma in patients with and without TAR*

	Nr. patients with TAR	*Nr. control patients*
Horizontal deceleration	49	31
Vertical deceleration	3	7
Combination of horizontal and vertical deceleration	9	1
Other, unknown	2	23
Total	63	62

The position of the patients at the time of the trauma is given in table III.4.
Of the 63 patients with TAR, 39 were initially admitted to one of the University Hospitals, 24 patients were referred later from community hospitals. Of the 62 control patients, 32 were initially admitted to one of the two University Hospitals, 30 were later referred from community hospitals. The time elapsed between the admission of the patients with TAR in the community and that in the University

Table III.4 Position of the patients with and without TAR at the time of the trauma

	Nr. patients with TAR	*Nr. control patients*
Automobile driver	33	12
,, passenger	13	5
Bicyclist	3	2
Motorbike- driver	4	3
Motorrider	2	3
Glider pilot	1	0
Pedestrian	0	1
Other/unknown	7	36
Total	63	62

Hospital was less than 24 hours for 17 patients (average 6 hours), 2 days for 5 patients, 7 days for one patient and 40 days for one patient. The average time elapsed between the trauma and the TAR patients arrival in the emergency room was short: one hour with a range of 0.5-6 hours. Other time factors will be presented in the appropriate chapters.

III.3. **Diagnosis**

III.3.1. *Clinical Symptoms*

III.3.1.1. *Patients with aortic rupture*

All the 63 patients with TAR were initially admitted to the emergency room of one of both University Hospitals or to a community hospital. The kind of trauma in most patients was known to the clinician (table III.3).
On the basis of the clinical symptoms, a diagnosis of TAR was only rarely suspected. The most frequent symptom was chest pain in 36/46 (78%) patients, which could be explained by fractured ribs (n = 28), sternum (n = 5), vertebrae (n = 6), clavicles or scapulae.
Dyspnea was present in 28 patients. Twenty five patients were in shock (systolic tension below 100 mm Hg.) at admission. More specific symptoms such as diminished or absent radial pulses were caused by concomitant brachio-cephalic lesions (n = 3); the more specific signs of TAR such as acute coarctation syndrome, paraple-

Table III.5 Clinical symptoms in 63 patients with TAR

	Nr. of patients	
Chest pain	36[1]/46[2]	78%
Dyspnea	21/45	47%
Hypotension left upper extremity	2/46	4%
Acute coarctation syndrome	5/47	12%
Anuria[3]	4/51	8%
Paraplegia[4]	3/63	5%
Systolic murmur	1/45	2%
Hoarseness	1/63	1%

[1] Nr. of patients in which the symptom was present.
[2] Nr. of patients in which this symptom could be evaluated.
[3] Caused by shock in three patients, in one patient associated with transection of the aorta.
[4] Caused by injury of the brain or spine.

gia, anuria or a systolic murmur over the sternal or interscapular area were infrequently present or not mentioned in the patient records. Back pain was present in one patient. Dysphagia as a sign of TAR was not seen in our patients. In one patient, hoarseness was the distinguishing symptom. Upper extremity hypertension was found in one patient. Table III.5 summarizes the clinical symptoms.
The clinical symptoms of our patients will be compared with those in the literature and discussed under heading III.4.2.

III.3.1.2. *Patients without aortic rupture*

All patients with blunt chest trauma, but without TAR were admitted to the emergency room of one of both University Hospitals or a community hospital. The type of trauma was known to the clinician in most cases (Table III.3). Clinical symptoms are summarized in table III.6.

Table III.6 Clinical symptoms in 62 patients with blunt chest trauma without TAR

	Nr. patients	
Chest pain	23/26	88%
Dyspnea	13/30	43%
Hypotension upper extremity*	2/38	5%
Acute coarctation syndrome	0/35	0%
Anuria	1/34	3%
Paraplegia	0/37	0%

* Due to trauma of the subclavian/axillary artery.

III.3.2. **Chest radiography**

III.3.2.1. *Patients with aortic rupture*

Apart from the clinical findings and a history of deceleration trauma, the chest radiograph played the most important role in suggesting to the radiologist a diagnosis of TAR.
For most patients (n = 59) anteroposterior chest radiographs were made in the supine position. Film focus distance was 100-115 cm. High KV technique (125 KV) and a grid were always used. Asymmetric projection was only a minor problem (4/60). The time elapsed between the first chest film and the trauma is given in table III.7. Chest films of 60 patients were available for analysis, in one patient an X-ray picture of the thoracic spine was used for analysis.

Table III.7 Elapsed time between the first chest film and the trauma in the patients with TAR

Time	*Nr. patients*	
0-1 Hour	13/63	21%
1-3 Hours	34/63	54%
3-6 Hours	3/63	5%
6-24 Hours	1/63	2%
24 Hours	1/63	2%
Unknown	11/63	17%

We analysed our chest roentgenograms (the first chest roentgenogram after admission) for 15 criteria related to mediastinal hematoma (table III.8), mentioned in the literature.

The following definitions were used:

1. Mediastinal width was considered abnormal when it exceeded 8 cm at the level of the aortic knob (Sturm and Marsh, 1976).
2. Mediastinal chest width (M/C) ratio is the quotient of chest width and mediastinal width and is determined at three levels:

MC I : at the level of the aortic knob

MC II : at the middle of the ascending aorta

MC III: at the middle of the descending thoracic aorta (Seltzer et al., 1981).

3. The trachea was considered displaced if the middle of the trachea extended to the right of the spinous process at Th 4/Th 5 (Barcia and Livoni, 1983).
4. Depression of the left main bronchus was subjectively evaluated.
5. A nasogastric tube was considered displaced if it extended to the right of the spinous process at the level of Th 4 (Gerlock et al., 1980).
6. Any aortic outline was considered abnormal if the contour was atypical or if the outline was not clearly and sharply visible (Barcia and Livoni, 1983).
7. Broadening of the paratracheal stripe was considered abnormal if it measured more than 5 mm (Woodring et al., 1982).
8. The right paraspinal line was considered displaced if it was separated from its normal close relationship with the lateral margin of the vertebral bodies (Peters and Gamsu, 1980).
9. The left paraspinal line was considered displaced if located at a distance greater than one half of the distance from the spine to the left margin of the descending thoracic aorta (Peters and Gamsu, 1980).

10, 11. First and second rib fractures are objective signs.

12, 13. Hemothorax or apical cap are objective signs.

14. Displacement of the superior vena cava and

15. Opacification of the clear space in the aortic pulmonary window were subjectively evaluated.

Table III.8, III.9 and Fig.III.2 summarize the chest X-ray findings in 63 patients with TAR.

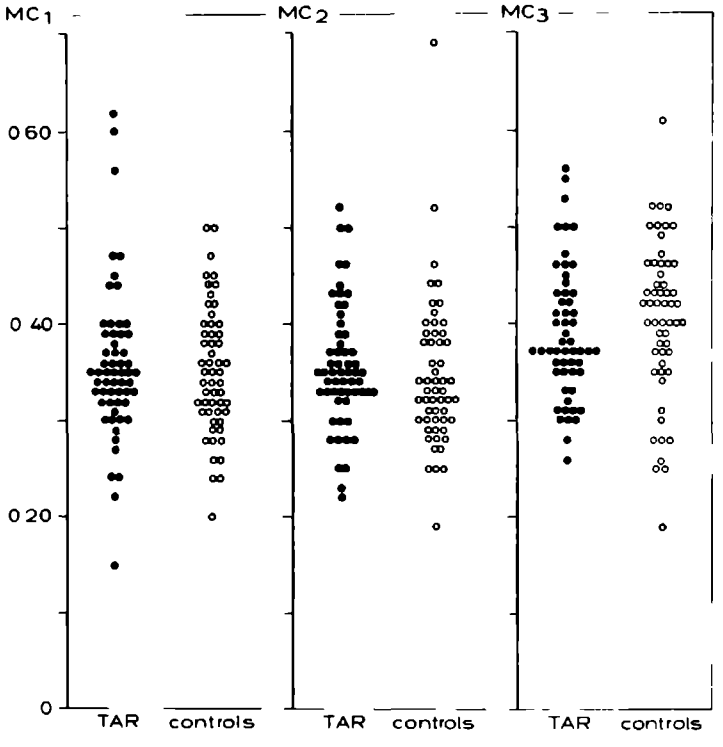


Figure III.2 Distribution of mediastinal chest width ratios at three levels (see text) in 63 patients with TAR and in 62 control patients.

III.3.2.2. *Patients without aortic rupture*

Table III.10 and III.11 and Fig.III.2 tabulate the chest X-ray findings in 62 patients without TAR.

Pulmonary contusion was found in 22/60 (36%) of the patients with TAR in comparison with 30/61 (47%) in the control patients. Diaphragmatic rupture was found in six patients with TAR but in none of the control patients. A pneumothorax was present in six patients with TAR in comparison with 16 patients of the control group. Fig. III.3-17 show examples of radiographic findings in our patients with TAR. Our chest X-ray findings will be discussed under heading III.4.3.

Table III.8 Chest X-ray findings in 63 patients with TAR

		Nr. patients	
1.	Mediastinal width		
	< 6 cm	0	
	> 6 < 8 cm	8/61	13%
	> 8 < 10 cm	24/61	39%
	> 10 cm	29/61	48%
2.	Mediastinal chest width ratio (see fig. III.2 and table III.9)		
3.	Tracheal displacement to the right	44/61	72%
4.	Downward displacement of the left main bronchus	45/61	74%
5.	Displacement of the nasogastric tube	17/29	59%
6.	Unsharp aortic outline	58/61	95%
7.	Broadening of the right paratracheal stripe	44/53	83%
8.	Displacement of the right paraspinal line	24/47	51%
9.	Displacement of the left paraspinal line	38/46	75%
10.	Fracture of the first or second rib	5/61	8%
11.	Other rib fractures	24/61	40%
12.	Left apical cap	42/61	68%
13.	Hemothorax	20/60	34%
14.	Displacement of the superior vena cava	29/58	50%
15.	Opacified pulmonary window	58/60	97%

Table III.9 Mediastinal chest width ratios in 63 patients with TAR*

	Average	*Range*
Mediastinal chest width ratio I	0.36	0.22-0.62
Mediastinal chest width ratio II	0.35	0.23-0.52
Mediastinal chest width ratio III	0.39	0.27-0.56

* See also fig. III.2.

Table III.10 Chest X-ray findings in 62 patients without TAR

		Nr. patients	
1.	Mediastinal width		
	< 6 cm	1/62	2%
	> 6 cm < 8 cm	18/62	29%
	> 8 cm < 10 cm	25/62	40%
	> 10 cm	18/62	29%
2.	Mediastinal chest width ratio (see fig III.2 and table III.11)		
3.	Tracheal displacement to the right	22/62	35%
4.	Downward displacement of the left main bronchus	10/62	16%
5.	Displacment of the nasogastric tube	4/36	11%
6.	Unsharp aortic outline	38/62	61%
7.	Broadening of the right paratracheal stripe	10/34	29%
8.	Displacement of right paraspinal line	4/51	8%
9.	Displacement of left paraspinal line	21/51	41%
10.	Fracture of the first or second rib	15/62	24%
11.	Other rib fractures	28/62	45%
12.	Left apical cap	33/61	54%
13.	Hemothorax	18/62	29%
14.	Displacement of the superior vena cava	19/62	31%
15.	Opacified pulmonary window	31/61	51%

Table III.11 Mediastinal chest width ratios in 62 patients without TAR*

	Average	*Range*
Mediastinal chest width ratio I	0.34	0.2-0.5
Mediastinal chest width ratio II	0.35	0.1-0.69
Mediastinal chest width ratio III	0.41	0.27-0.61

* See also fig. III.2.

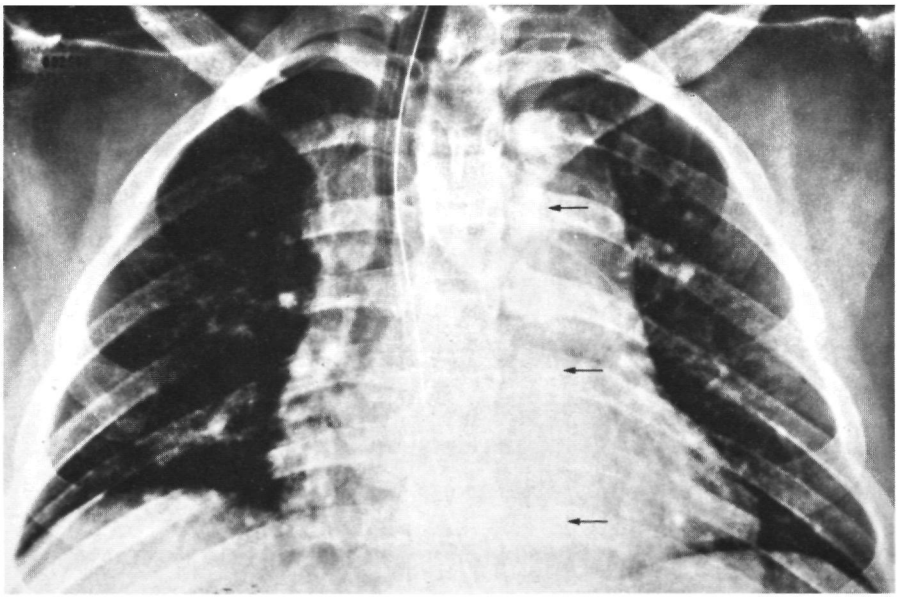


Figure III.3 Typical presentation of a patient with TAR (male, 23 years, car passenger, uneventful recovery after operation). Chest X-ray: trachea and nasogastric tube displaced to the right. Widening of the mediastinal shadow (12 cm). Caudal displacement of the left main bronchus. Widened left paraspinal line (arrows).

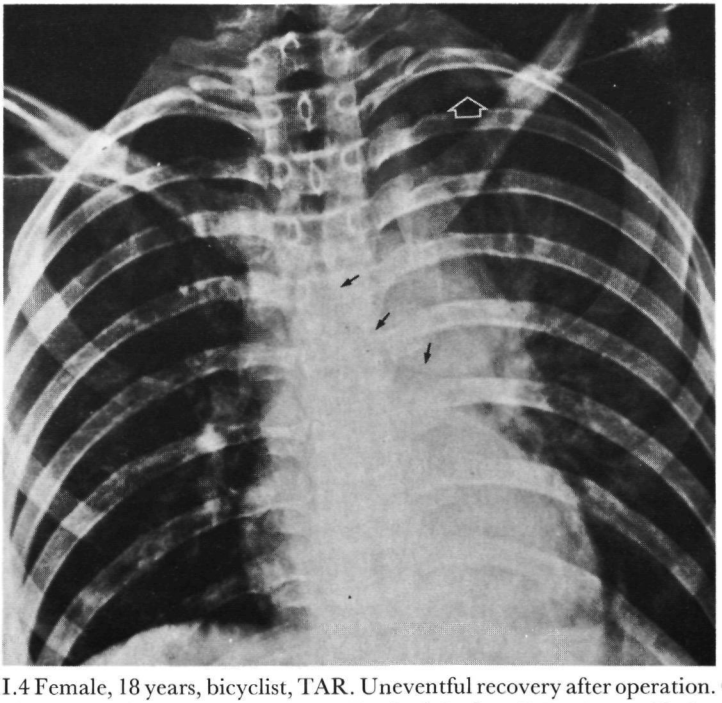


Figure III.4 Female, 18 years, bicyclist, TAR. Uneventful recovery after operation. Chest X-ray: widening and unsharp contour of the mediastinal shadow (8.5 cm), opacified pulmonary window with discrete displacement of left main bronchus (arrows). Apical cap (open arrow).

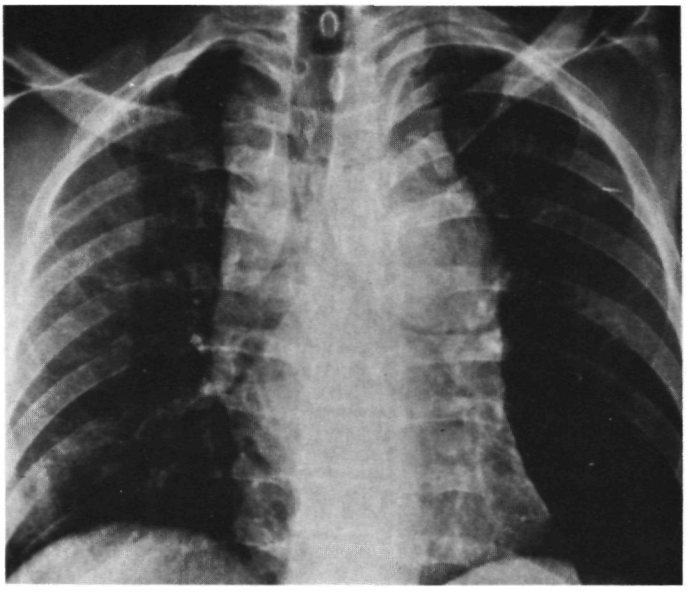


Figure III.5 Male, 35 years, car-driver, TAR, uneventful recovery after operation. Chest X-ray: diffuse widening of the mediastinal shadow (12 cm) with sharp contours. Aortic knob not visible. Displacement of the trachea to the right. Widened paratracheal stripe. Normal paraspinal lines. Angiography: rupture of the aorta between the innominate artery and the right carotid artery (see also Fig.III.19b; same patient).

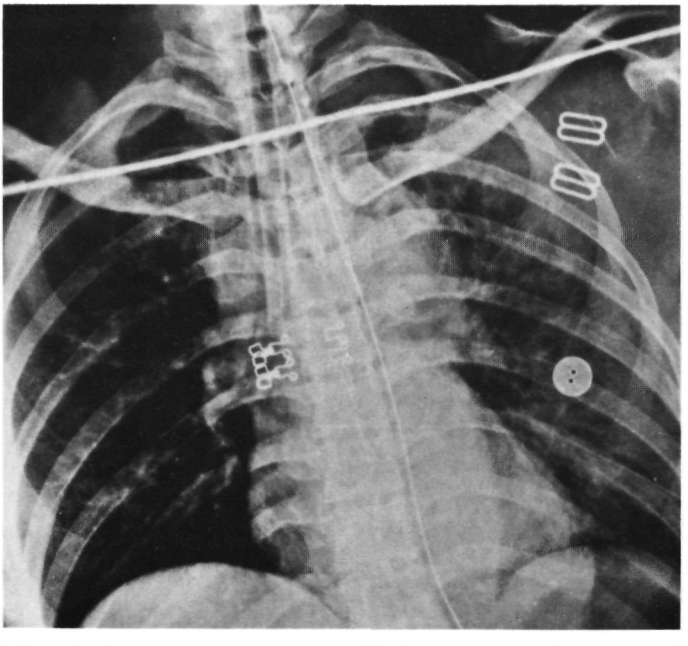


Figure III.6 Female, 30 years, car-passenger, TAR. Death on the 6th postoperative day due to sepsis. Chest X-ray: tracheal displacement to the right and normal position of the nasogastric tube. Widening of the mediastinal shadow (10 cm). Unsharp aortic outline. Widened paratracheal stripe.

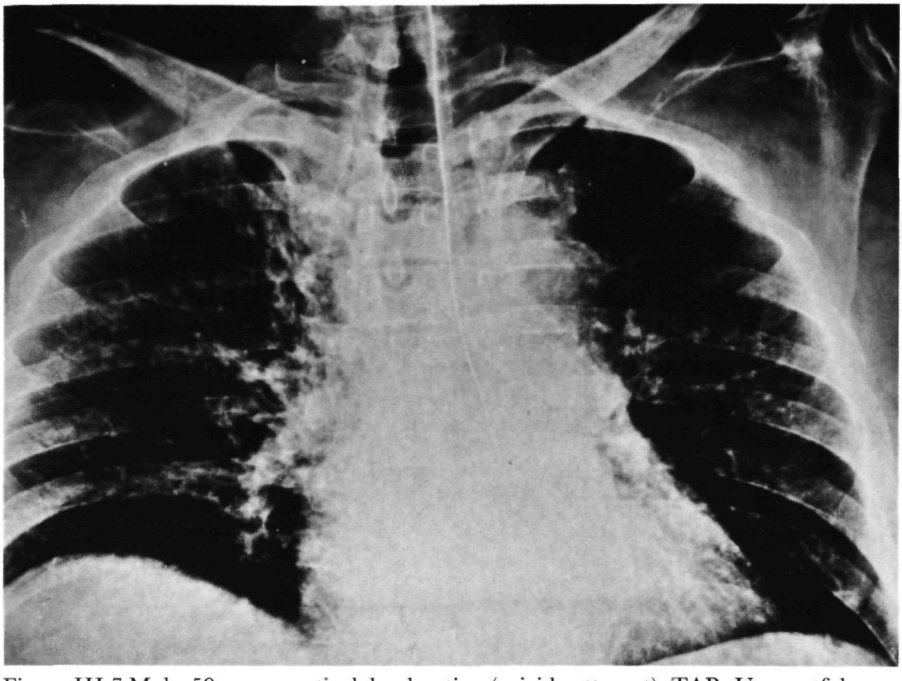


Figure III.7 Male, 59 years, vertical deceleration (suicide attempt), TAR. Uneventful recovery after operation. Chest X-ray: normal position of trachea and nasogastric tube. Widening of the mediastinal shadow (10 cm). Unsharp aortic outline. Opacified pulmonary window.

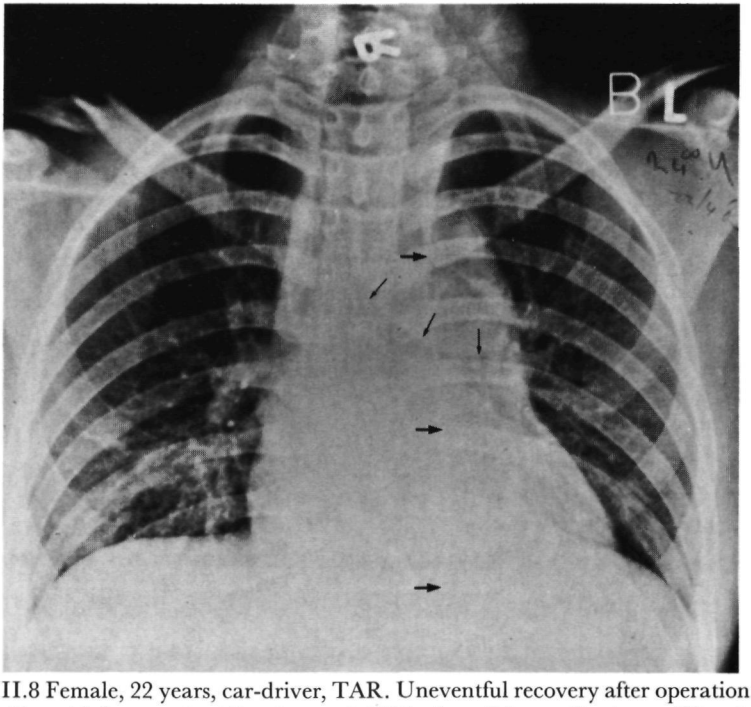


Figure III.8 Female, 22 years, car-driver, TAR. Uneventful recovery after operation. Chest X-ray: widened left paraspinal line (arrows). Widening of the mediastinum (10 cm). Slight displacement of the trachea and left main bronchus (arrows), widened paratracheal line.

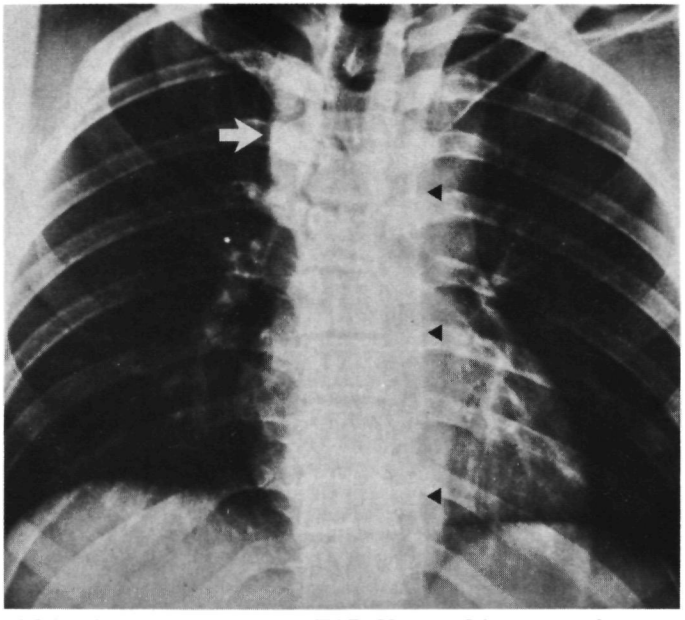


Figure III.9 Male, 18 years, car-passenger, TAR. Uneventful recovery after operation. Chest X-ray: widened left paraspinal line (triangles). Widened paratracheal line (white arrow). Widening of the mediastinal shadow (9 cm) and unsharp aortic outline.

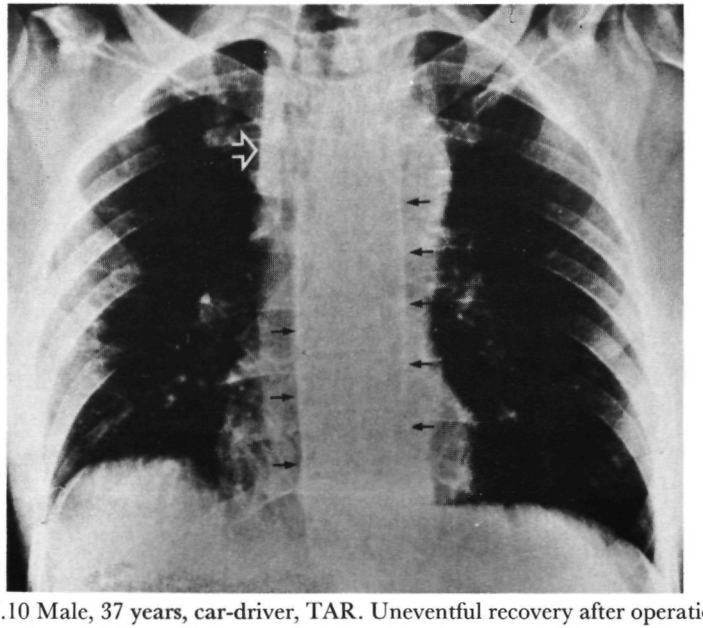
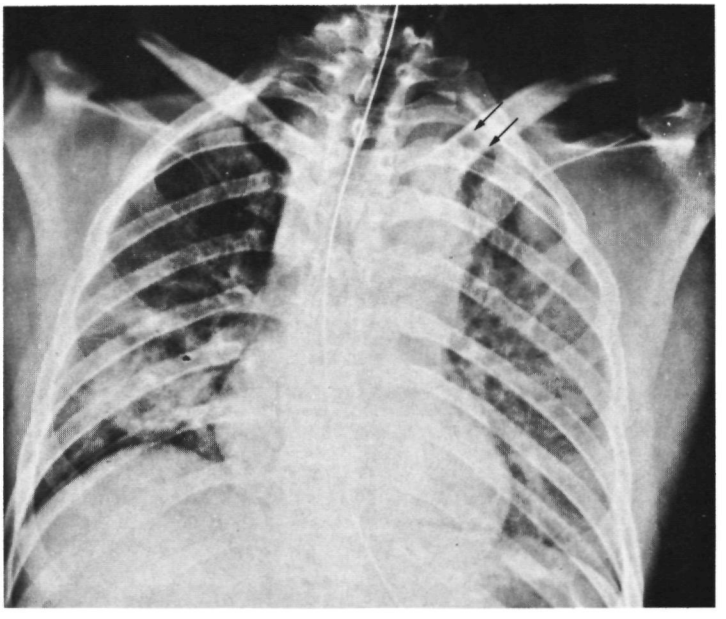


Figure III.10 Male, 37 years, car-driver, TAR. Uneventful recovery after operation. Chest X-ray: widening of right and left paraspinal lines (black arrows). Widening of paratracheal stripe (white arrow). Note also tracheal displacement and widening of the mediastinal shadow (10 cm) with smooth contours.



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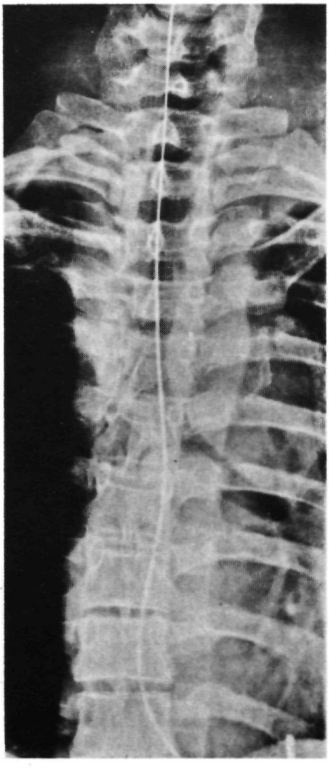

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Figure III.11 Male, 29 years, car-driver, TAR. Uneventful recovery after operation. a. Chest X-ray and b. Thoracic spine. Normal position of trachea and nasogastric tube. Widening of the mediastinal shadow (9 cm). Opacified pulmonary window. Apical cap (arrows). Lung contusion of right lower lung. Widened right paratracheal stripe.

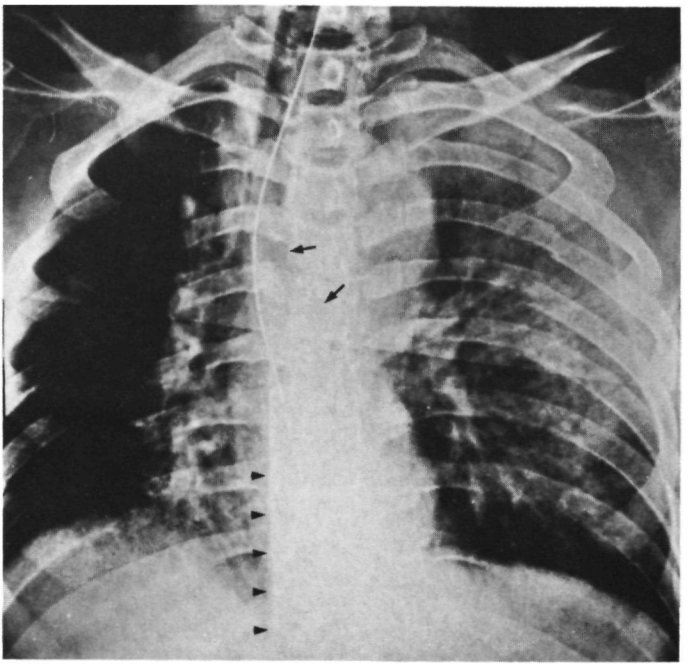


Figure III.12 Male, 18 years, vertical deceleration, TAR. Death on the 9th postoperative day due to cerebral injury. Chest X-ray: widening of the right paraspinal line (triangles). Note also displacement of the trachea and nasogastric tube (arrows). Depressed left main bronchus. Widened mediastinal shadow (12 cm). Unsharp aortic contour. Pulmonary contusion of the left side with multiple fractures.

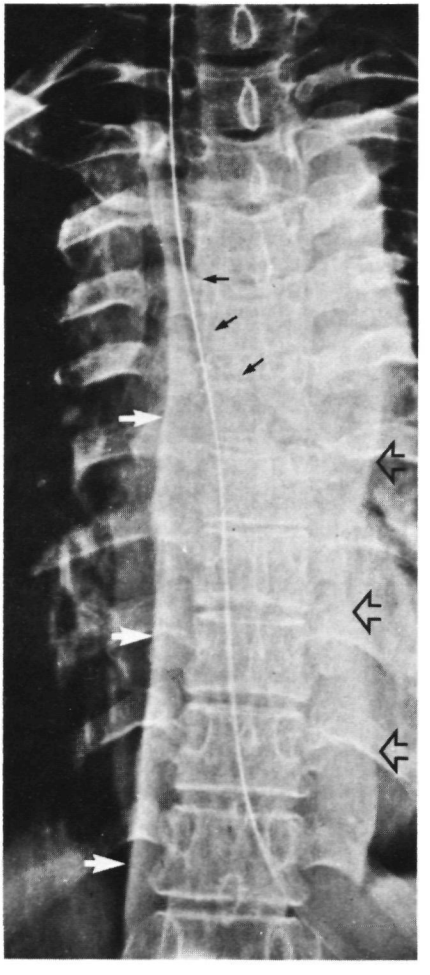


Figure III.13 Female, 47 years, car-driver, TAR. Death during celiotomy due to massive bleeding. Chest X-ray: broadening of left (open black arrows) and right (white arrows) paraspinal lines. Note also displaced trachea (black arrows) and nasogastric tube. Widening of the mediastinal shadow (11 cm). Widened right paratracheal stripe.

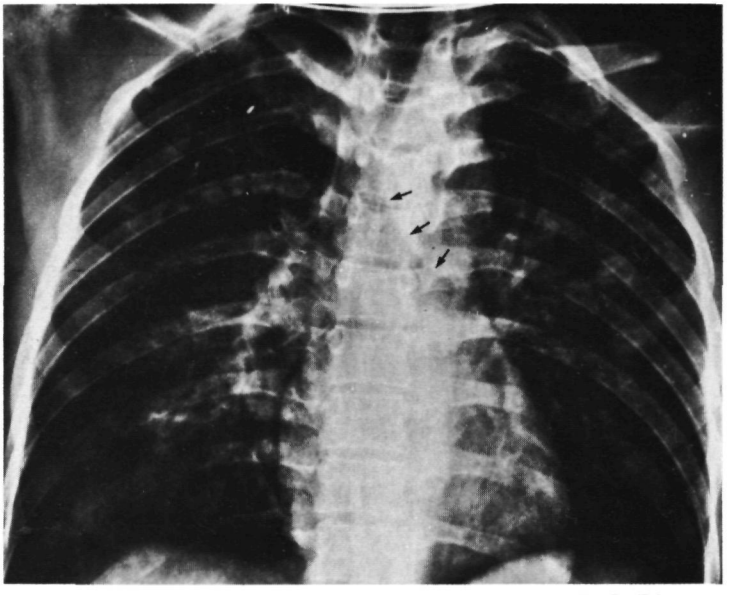


Figure III.14 Male, 21 years, car-driver. Death due to unrecognised TAR. Diagnosis of TAR at autopsy. Chest X-ray: mediastinal width 7.5 cm. Sharp aortic outline. Only slight displacement of the trachea and left main bronchus (arrows).

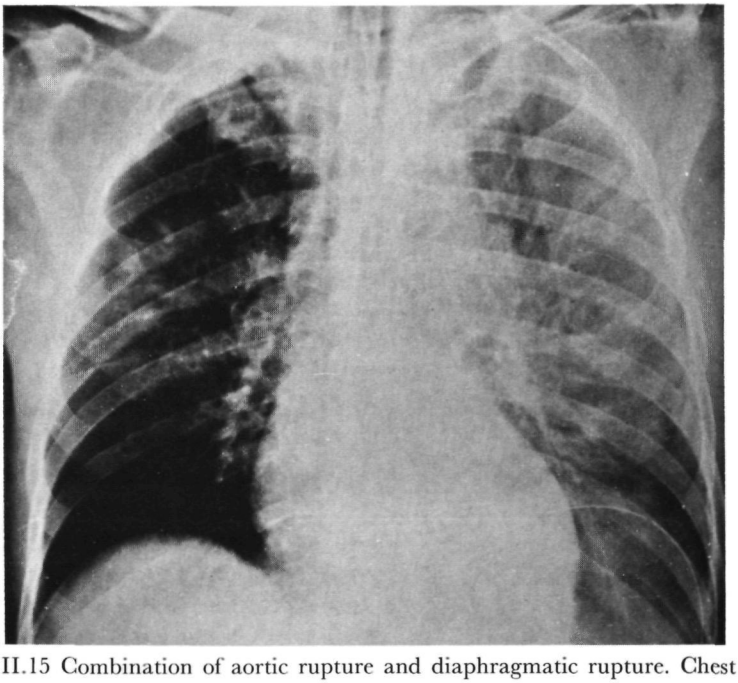
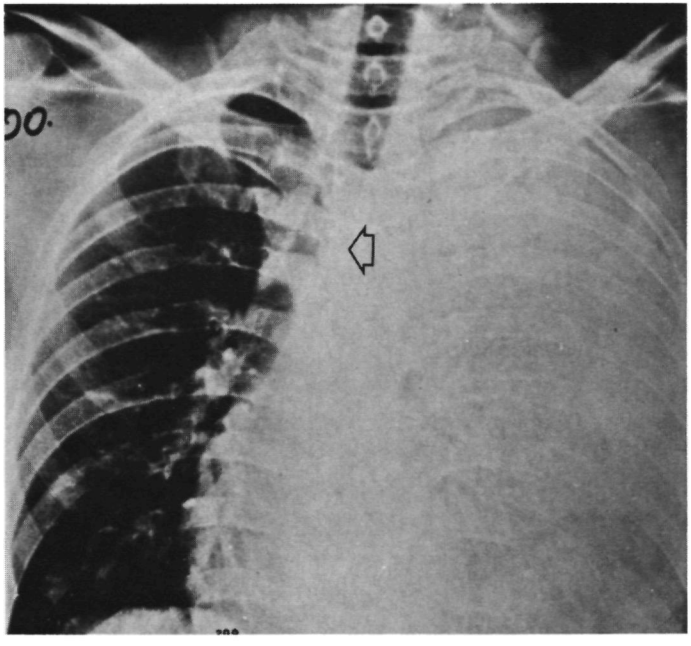


Figure III.15 Combination of aortic rupture and diaphragmatic rupture. Chest X-ray: widening of the mediastinal shadow (9 cm) and unsharp aortic contour. Contusion of the left lung. Opacified pulmonary window. No displacement of trachea or nasogastric tube. Left hemi-diaphragm not visible. Patient (male, 60 years, car-driver) died from unrecognised TAR, while diaphragmatic repair was performed.



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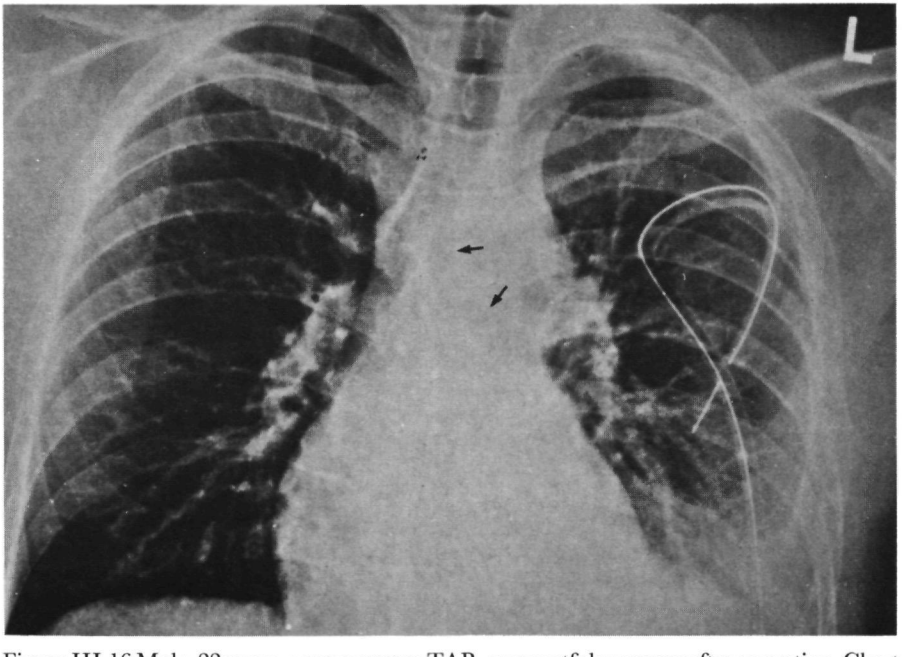

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Figure III.16 Male, 22 years, car passenger, TAR, uneventful recovery after operation. Chest X-rays: (a) before drainage of a left sided hemothorax. Displacement of the trachea to the right (arrow). Mediastinal contours cannot be evaluated. (b) after drainage of the left sided hemothorax the mediastinum can be evaluated. Abnormal unsharp contour of the proximal descending aorta; impression on the trachea and left main brochus (arrows); opacified pulmonary window. Angiography: TAR.

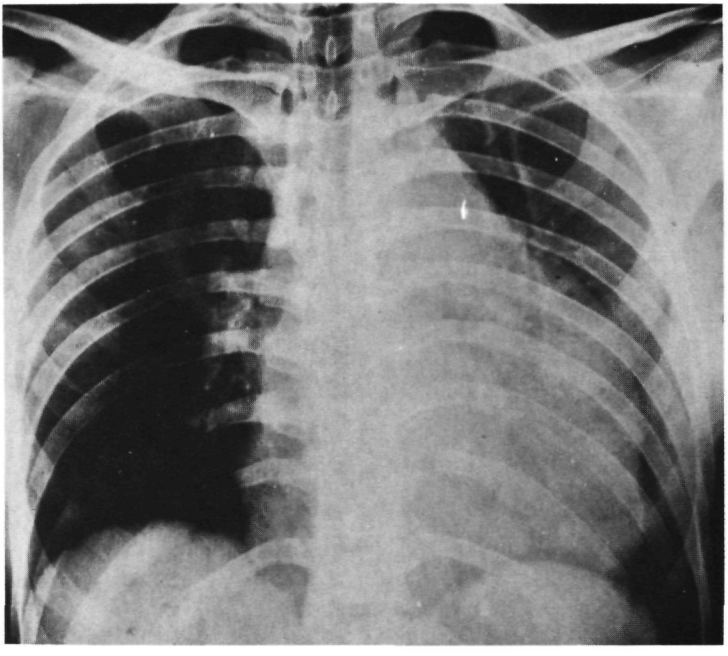


Figure III.17 Male, 24 years, car passenger, TAR, uneventful recovery. Combination of aortic rupture and cardiac pathology. Chest X-ray: abnormal aortic contour at the level of the aortic knob. Widening of the mediastinum. Normal position of the trachea. Massive enlargement of the cardiac silhouette. Angiography: TAR. At operation hemopericardium due to rupture of the vena cava was found.

III.3.3. **Angiography**

Angiography was performed as soon as possible after the suspicion of aortic rupture was raised. In nearly all cases this suspicion grew from the chest X-rays. In five patients, no angiogram was obtained: in two patients because TAR was not suspected, in three cases the patients died before angiography could be performed. As

Table III.12 The elapsed time between trauma and angiography in 52 patients with TAR

Time	*Nr. of patients*	
0- 1 Hour	0	0%
1- 3 Hours	17/52	33%
3- 6 Hours	12/52	23%
6 Hours	7/52	13%
24 Hours	10/52	19%
Unknown	6/52	12%

obvious, no angiograms were performed in the six patients with TAR in whom the aneurysm was not detected at first admission.
Table III.12 gives information with regard to the time interval between the trauma and the angiography.

III.3.3.1. *Technique of angiography*

In most instances (table III.13) angiography was performed by the femoral route. In one case it was necessary to use the brachial approach because of the absence of the femoral pulses. In two cases a complementary investigation via the brachial route was necessary because the catheter could not be passed through the traumatised segment of the aorta from below. Sometimes a patient with an angiogram from elsewhere was presented (n = 5).
The angiographic technique is presented in table III.13.

Table III.13 Angiographic pathway in 52 patients with TAR

	Nr. patients
Femoral	49
Brachial	1
Brachial and femoral	2

In most patients a 7.1 French pigtail catheter with 12 side holes (Cook) was positioned in the ascending aorta, just before the take off the brachio-cephalic vessels. Urografin (76%) 30 cc/sec was injected for two seconds (Nijmegen) or Telebrix 30, 25 cc/sec for three seconds (Groningen). Filming was performed with an AOT film changer (Nijmegen), 3 films per second for three seconds, 2 films per second for two seconds or with a 70 or 100 mm camera mounted on a C-arm equipment that had a film program of 2 pictures/sec. for 6 seconds (Groningen). Preferably the 60 to 70 degree left anterior oblique projection (n = 39) was used as in this position the aortic arch is unfolded and evaluation of the proximal descending aorta is optimised. Sometimes the LAO projection could not be obtained due to other traumatic skeletal injuries and the AP projection then was used (n = 13). In a few cases, both AP and LAO projections were used. In general, angiography took less than 30 minutes in Groningen because patients could be investigated on the same table with a C-arm equipment. In Nijmegen additional time (15 - 30 minutes) was required because the patients had to be transported to an adjacent angiographic room. The angiograms of all the 52 patients with TAR in whom angiography was performed were available for review.

III.3.3.2. *Angiographic findings*

Table III.14 shows the angiographic findings in 52 patients with TAR

Table III.14 Angiographic findings in 52 patients with TAR

	Nr. patients
False aneurysm	45
Dissection	2
Intimal tear	1
Transection	2
Contrast extravasation	1 (1)
Multiple aneurysms	(1)
Brachio-cephalic vessel injury	(3)
Other findings	1

() Double counting

Table III.15 tabulates the location of the aortic rupture in 57 patients and Fig. III.18-22 shows examples of angiographic findings in our patients.

Table III.15 Localisation of the aortic rupture in 57 patients

	Nr. patients
Ascending aorta	0
Aortic arch	4
Isthmus	48*
Descending thoracic aorta	5 (1)
Combination of localisations	(1)

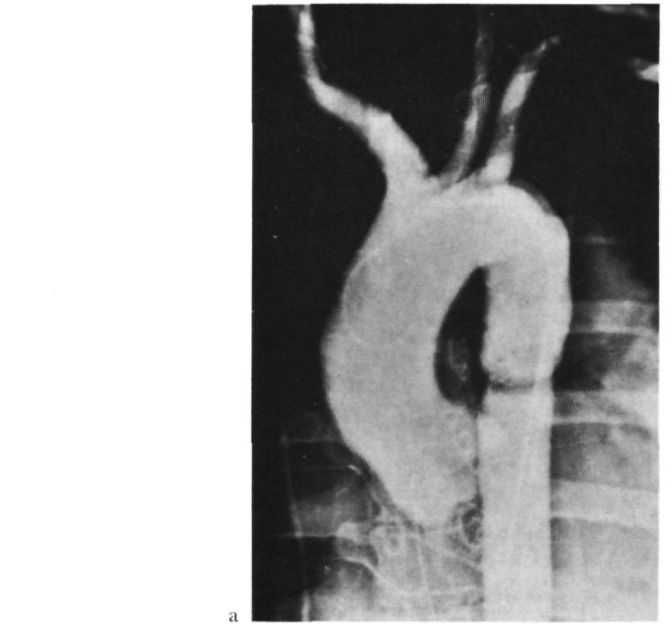
* In five patients found at autopsy.
( ) Double counting

In three patients, lesions of the brachio-cephalic vessels were present. In two a false aneurysm of the left subclavian artery was detected and one patient showed a dissection of the left carotid artery.
In 18/60 (30%) of the patients, angiography of the abdominal aorta and vessels of the liver, spleen and kidney were performed in the same session.
Our angiographic findings will be discussed under heading III.4.4.

III.3.3.3. *Complications of angiography*

No complications were encoutered except in one case in which extravasation of contrast occurred because the pigtail catheter had entered the false aneurysm (fig.

a



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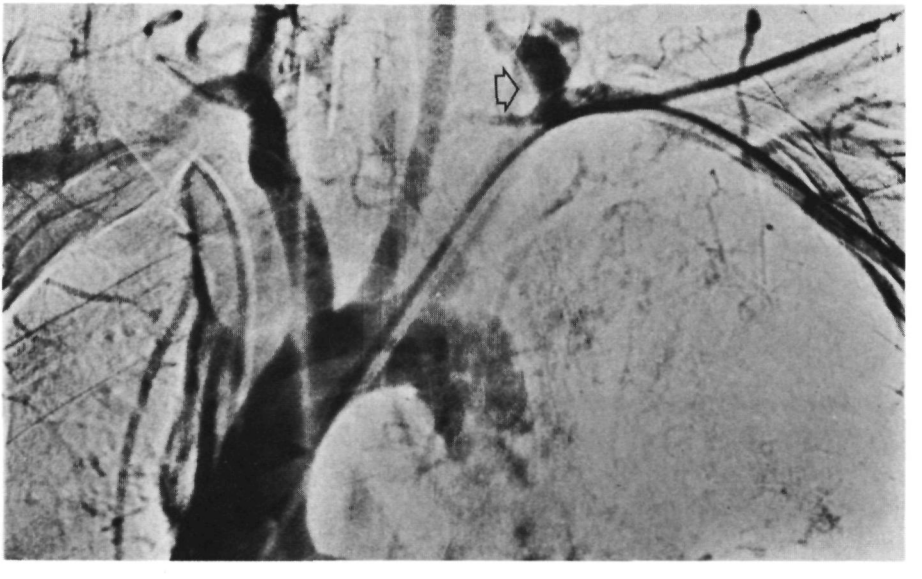
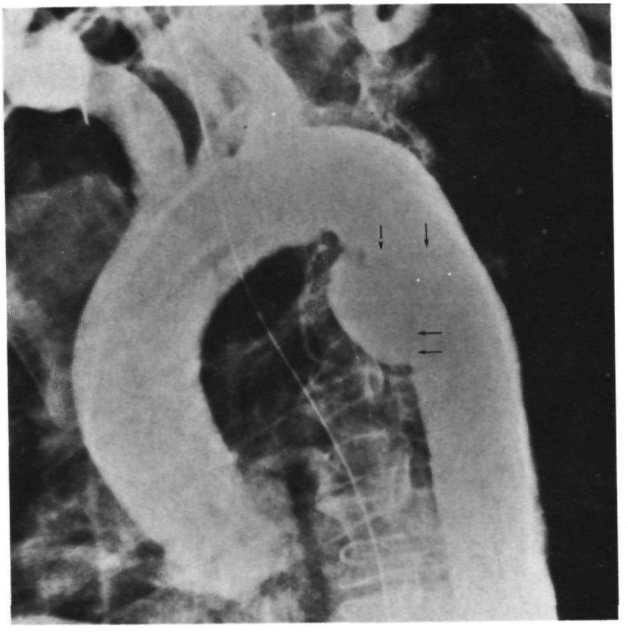


Figure III.18 Examples of various forms of traumatic aortic ruptures. a. Circumferential tear (atypical localised rupture in the descending aorta). b. Transection of all layers of the aortic wall with occlusion of the aortic lumen. Note small false aneurysm at the origin of the left subclavian artery (open arrow). c. Localisation of the false aneurysm at the inner curvature of the aorta. Note intimal flaps (arrows). d. Localisation of the false aneurysm at the outer curvature of the aorta. e. Local dissection without false aneurysm (white arrows).



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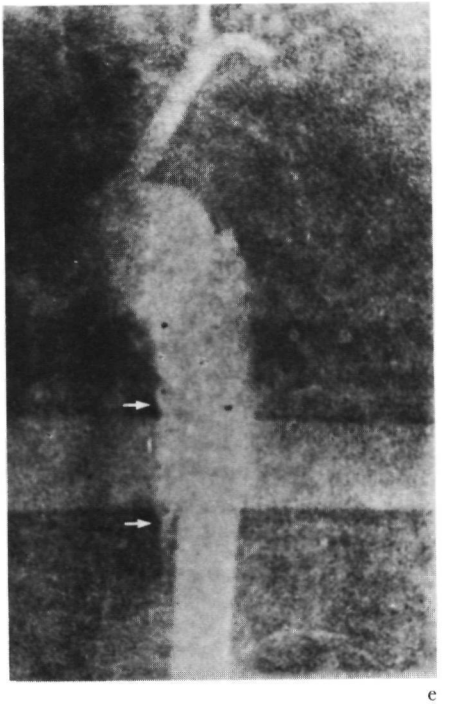

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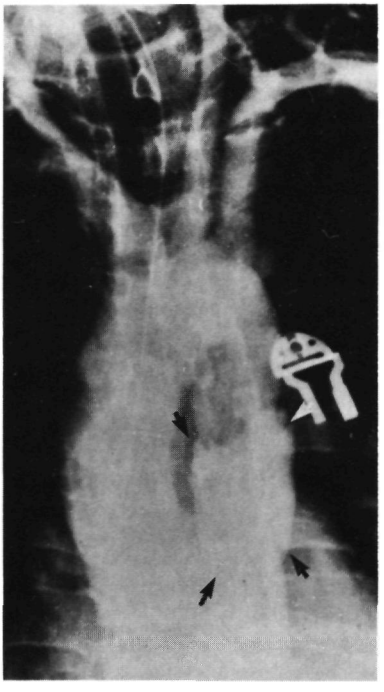
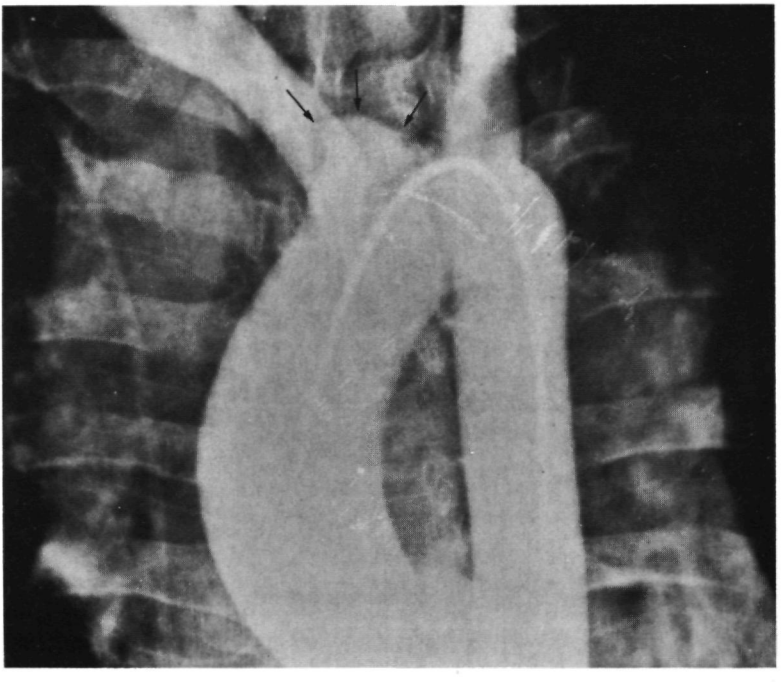


a



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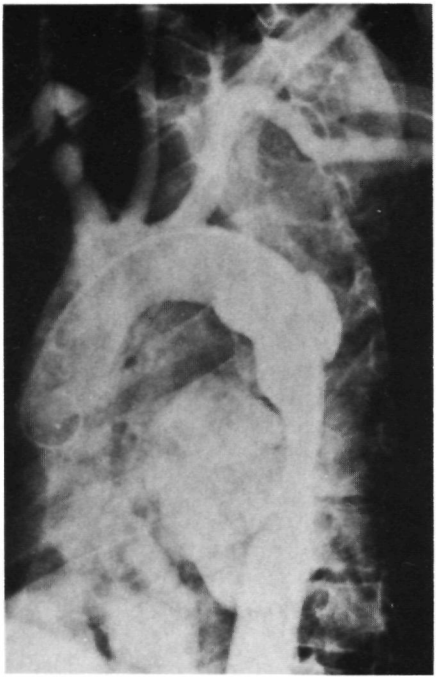

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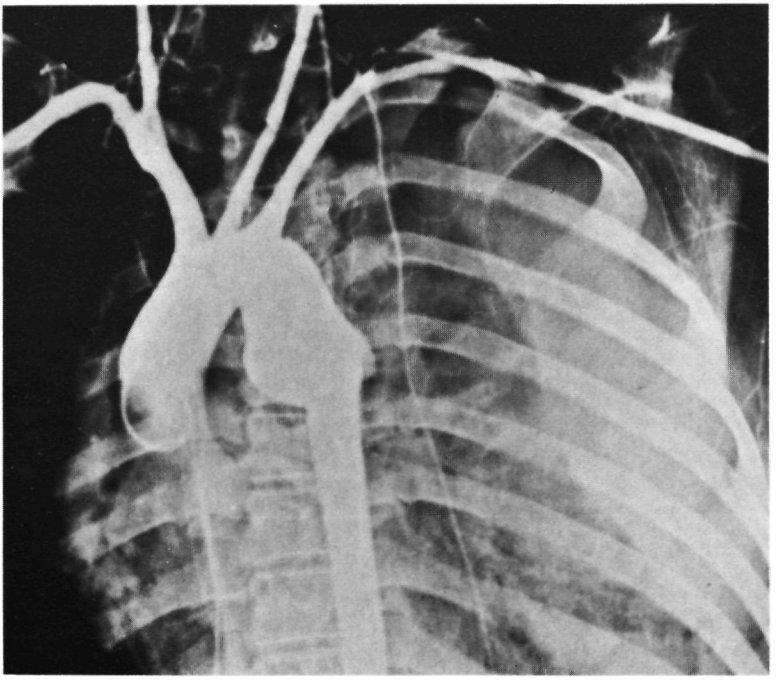
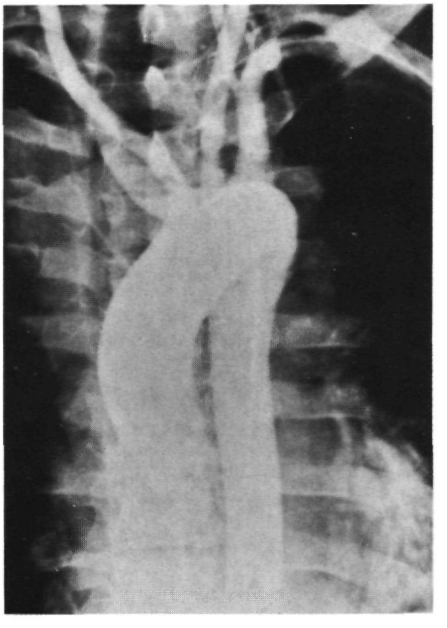


d

Figure III.19 Examples of different localisations of traumatic aortic rupture.
a. Localisation in the proximal descending thoracic aorta (arrows). b. Localisation in the aortic arch between the innominate artery and the left carotid artery (arrows). c. Multiple ruptures; one in the proximal descending aorta at the outer curvature: the other at the inner curvature of the mid descending aorta. d. Localisation at the isthmus.



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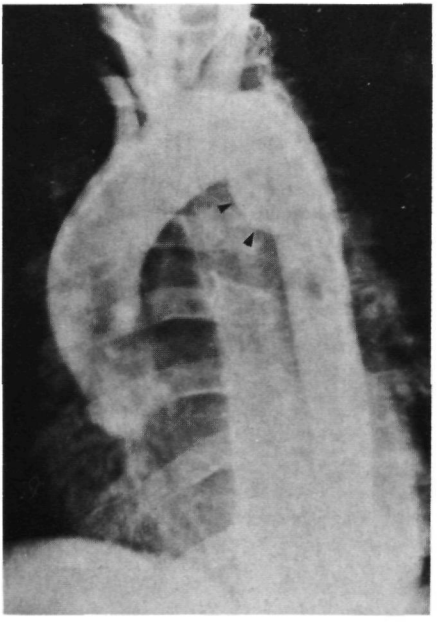
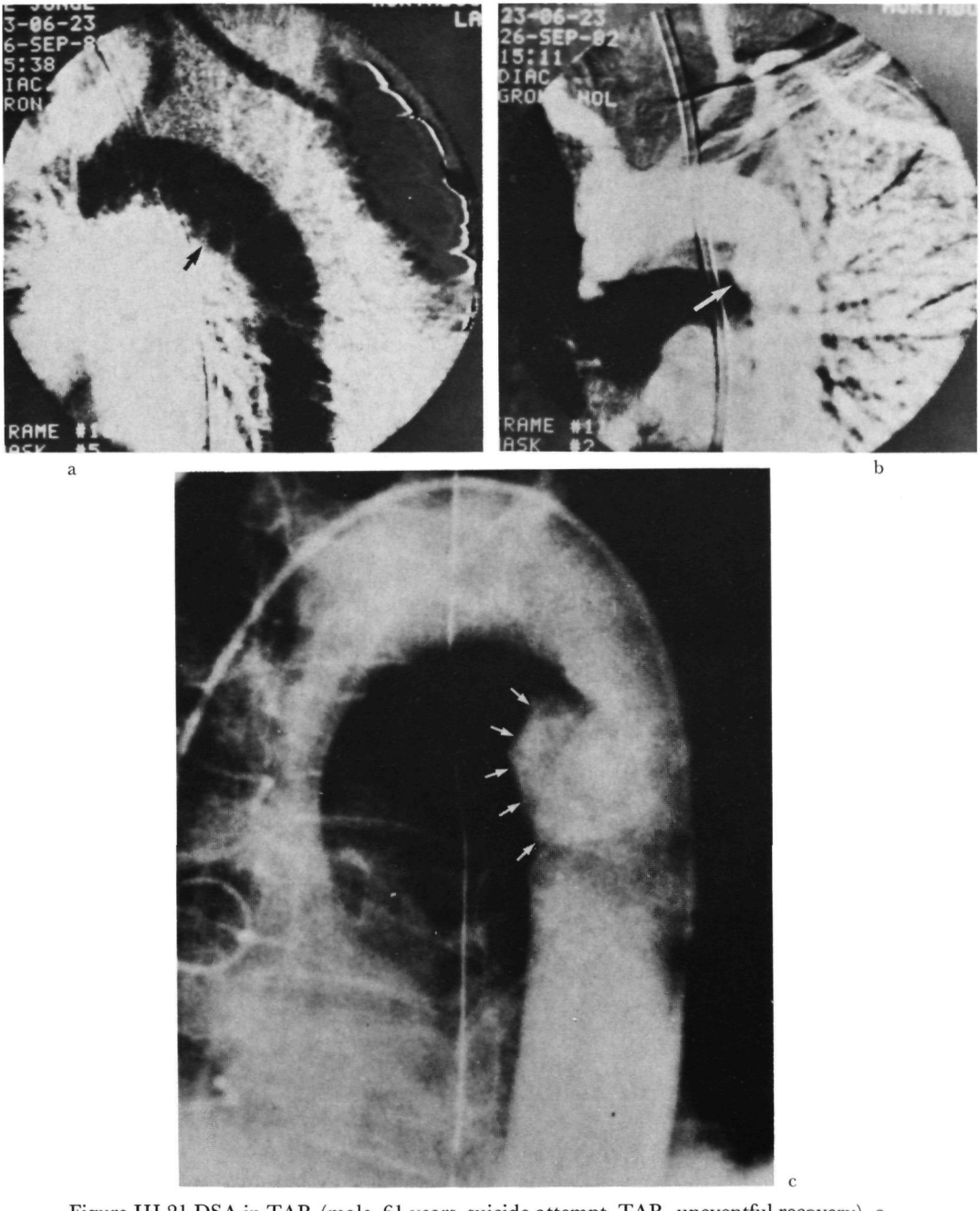


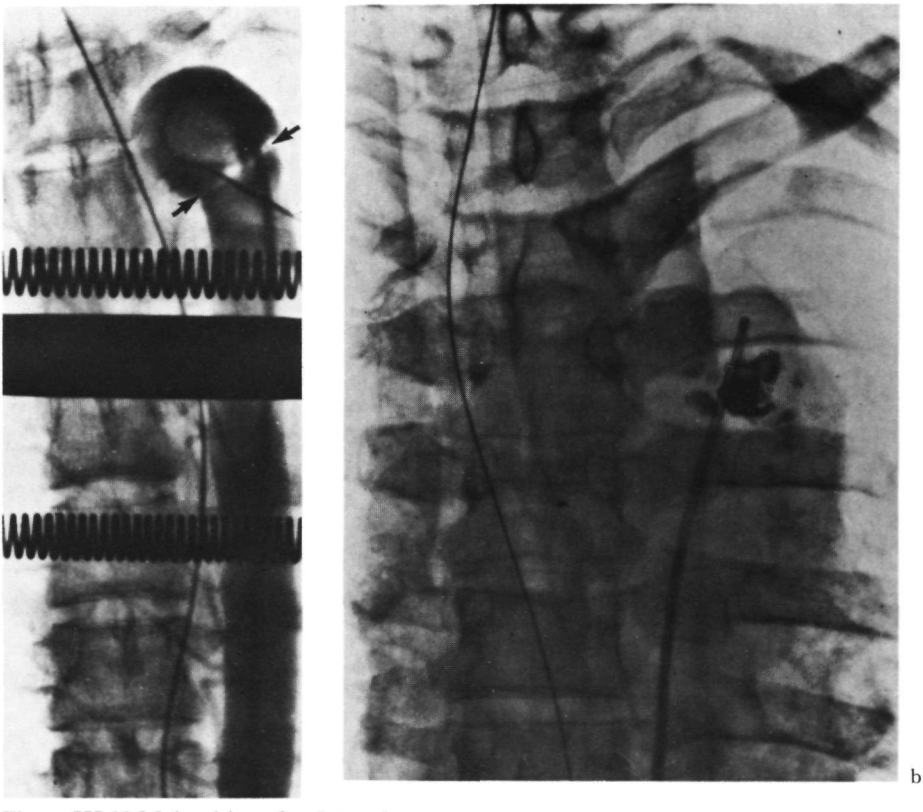
b

Figure III.20 Importance of LAO projection for the detection of TAR. a. AP projection: arteria lusoria; false aneurysm at the isthmus barely visible. b. LAO projection: good visualisation of a false aneurysm at the isthmus (triangles).



a b c

Figure III.21 DSA in TAR (male, 61 years, suicide attempt, TAR, uneventful recovery). a and b. Intravenous DSA aortic arch study: interpreted as negative. Retrospectively, suspicion for local false aneurysm (arrows).
c. Conventional angiography: typical false aneurysm at the isthmus (arrows).



a b

Figure III.22 Malposition of catheter tip.
a. Pigtail catheter in false aneurysm: contrast injection without sequelae. Note intimal flap (arrows). b. Tip of the catheter in false aneurysm: probably perforation of aortic wall. Extravasation of contrast medium in mediastinal tissues during test injection. No angiography performed.

III. 22.b). No angiogram was obtained in this patient. The patient died peroperatively due to bleeding from liver and spleen lacerations. In four patients, it was not possible to pass the area of the rupture because of infolding of the intima and subsequent occlusion of the aorta. In two of these, a complementary angiogram was performed via the brachial route.

III.4 **Discussion**

III.4.1. *Own patients*

Our group of patients with TAR (n = 63) is relatively large compared with other series from the literature, table III.16.

Table III.16 Number of patients with TAR where angiography was performed

Year	*Author*	*Nr. patients*
1971	DeMeules et al.	15
1975	Appelbaum et al.	25
1976	Kirsh et al.	38
1978	Ayella	67
1980	Motin et al.	36
1981	Akins et al.	44
1981	Fisher et al.	47
1983	Barcia and Livoni	17
1984	Own series	52

The majority of our patients (76%) were male, 24% were female. Most patients in both groups were of relatively young age (see table III.1), which is in accordance with the literature: most patients with TAR were in the younger age group. Age varied from 17 to 76 years, with an average of 36 years. The time between the trauma and arrival at the emergency room of a hospital was short, in average one hour; this is due to the relative short distances in our country and the organisation of the ambulance services.

As in most other reported series, automobile accidents causing deceleration and impact were the most important cause of TAR (73%). Typical for The Netherlands are the seven bicyclist- and motorbike accidents.

39/63 (62%)of the patients were admitted initially to the University Hospitals, 24/63 (38%) patients were admitted to a community hospital and later referred, 17 patients less than 24 hours later, five patients within two days, one patient after 7 days and another patient 40 days later. The time interval between the trauma and the operation was in general shorter for patients initially admitted to the University Hospitals than for those referred from other hospitals. Several patients from the control group were referred to one of the University Hospitals, to exclude a TAR by means of angiography.

III.4.2. *Clinical symptoms*

1. *Chest pain*

Chest pain was present in 36/46 (78%) patients with TAR. This is quite conceivable as many patients also had rib fractures 28/61 (46%); 5 patients had documented sternal fracture. The real presence of sternal fractures probably is much higher, as many patients had a steering-wheel injury. In general, in Nijmegen no radiographs of the sternum or a lateral chest X-ray were obtained. Five patients had fractures of

the thoracic spine. All these different kinds of fractures can explain chest pain. Differences in the frequency of chest pain in patients with and without TAR could not be found 36/46 (78%) versus 23/26 (88%), ($p > 0.05$). This makes chest pain a frequent but aspecific symptom for TAR.

2. *Dyspnea*

This symptom was present in 21/45 (47%) patients with TAR and in 13/30 (43%) of the control patients ($p > 0.05$). Most often dyspnea could be explained by concomitant pulmonary contusion. This was present in 22 of 60 (37%) patients with TAR; another reason was a pneumothorax ($n = 6$) or hemothorax ($n = 20$).
Dyspnea is a frequent but aspecific symptom both in TAR patients and in patients with blunt chest trauma without TAR.

3. *Dysphagia*

This symptom, reported by McBurney and Vaughan (1961) was not encountered in our patients with TAR.

4. *Acute coarctation syndrome*

The acute coarctation syndrome (A.Co.S.) was present in 5 of 47 patients (11%). In three of these it was associated with angiographically or surgically proven complete transection of the aorta, separating the two ends of the aortic wall, varying from 5 to 8 cm.
One patient showed a strong retraction of intima and media. Separation and retraction of the aortic wall was probably the cause of the A.Co.S. In one patient with A.Co.S. an arteriography was performed via the left brachial artery; in another patient the transected aorta could not be retrogradely passed. In one patient with A.Co.S. a false aneurysm was found at the typical site that is the aortic isthmus; the cause of his A.Co.S. was not clear. Of the 11 patients with documented complete transection of the aorta (Table V.6), only four showed the A.Co.S. Transection of the aorta seems to have a positive correlation with A.Co.S., probably by severe damage which leads to retraction of intima and media resulting in partial occlusion of the aortic lumen. A.Co.S. is however not inevitable with transection of the aorta, which was more frequent than A.Co.S.
Subadventitial hematoma or dissection as a cause of A.Co.S. was not seen in our patients.
Frequency of A.Co.S. in our patients with TAR (11%) is lower than that reported by Symbas (1972): 30%; Kirsh et al.(1976): 37%; Motin et al.(1980): 66%, but higher when compared with the reports of Appelbaum et al. (1975): 5%, Fisher et al.(1981): 4%. The reason for these discrepancies is not clear. In patients with an acute coarctation syndrome, a strong indication of an acute aortic rupture exists. In these patients it is important to take the angiographic pathway in consideration, as often complete transection of the aorta will be present and it will then be impossible

to pass the rupture from below. A brachial or axillary approach must be the first choice in these patients. Since A.Co.S. is a specific clinical symptom for TAR, it was not present in the control group.

5. *Paraplegia/anuria*

Paraplegia was present in 3/63 (5%) patients with TAR on admission. In one patient a cauda syndrome was present due to a fractured lumbar vertebra. In the other patient an epidural hematoma was probably the reason for the paraplegia. The third patient showed a fracture of the cervical spine at C4 and C5.The paraplegia was the result of these fractures or of the epidural hematoma. Paraplegia caused by complete aortic transection or in combination with a A.Co.S. was not encountered. Our percentage of preoperative paraplegia (5%) is lower than that in the literature (table II.7), where in large series percentages as high as 10 - 13% are reported. No patients with paraplegia were seen in the conrol group. Four patients with TAR were anuric on admission. Three of these patients were in shock, the other patient had a complete transection and interruption of the aortic lumen. One patient in the control group showing anuria was in shock.

6. *Upper extremity hypertension*

One of the patients showed upper extremity hypertension, which is contrary to the reports of Kirsh et al. (1976), Turney et al.(1977), who found this symptoms in 43, respectively 72% of their patients. We are unable to explain this rather large discrepancy.

7. *Systolic murmur*

A systolic murmur was not recorded in any of our patients with TAR, although it is quite conceivable that a systolic murmur was present, but not noticed.
Symbas (1972), Kirsh et al.(1976), Motin et al.(1980) and Barcia and Livoni (1983) noted a precordial or subscapular systolic murmur in 12 - 60% of their patients. One of the disadvantages of a retrospective study such as this one, is that it is not possible to properly evaluate the presence and frequency of these clinical signs.

8. *Other symptoms*

One patient developed in the week after a blunt chest trauma symptoms of hoarseness due to paralysis of the left recurrent nerve. This symptom was the reason for angiography which subsequently disclosed a traumatic aneurysm of the thoracic aorta.
Diminished brachial or radial pulsation were seen in 2 patients with TAR. These patients showed concomitant lesions of the left subclavian artery. These diminished pulsations can be a hint for a more serious aortic tear.
After reviewing the clinical symptoms of our patients, it can be stated that they were in general aspecific with no significant difference present in comparison with a

group of chest trauma patients without aortic rupture. Only the acute coarctation syndrome in 5 patients and hoarseness in one patient were rather specific indicators for TAR.

III.4.3. *Chest radiography*

Contrary to the opinion of Ayella et al. (1971), who advocated the erect chest roentgenogram in blunt chest trauma patients, we believe that a supine chest radiograph of trauma patients will provide adequate information concerning the mediastinum. Furthermore, it is generally impossible to obtain erect chest roentgenograms with these patients. All the chest roentgenograms of our patients with one exception were made in the supine position. Asymmetric positioning was encountered in only four patients and was a minor problem. We will discuss the findings on the chest roentgenograms separately.

1. *Mediastinal widening*

None of our patients with TAR had a mediastinal width (MW) less than 6 cm; in 8 patients the MW was between 6 and 8 cm, in 24 patients between 8 and 10 cm and in 29 patients more than 10 cm. The average MW was 9.7 cm (table III.17), comparable with the 9.7 cm in the series of Sturm and Marsh (1976) and the 9.8 cm in the series of Barcia and Livoni (1983). Woodring et al. (1982) reported however 8.2 cm.

Table III.17 Mediastinal widening in our patients with and without TAR.

Mediastinal width	<6 cm	6-8 cm	>8cm	> 10 cm	Average
TAR (61 patients)		8 (13%)	53 (87%)	29 (48%)	9.7 cm
Controls (62 patients)	1(2%)	18 (29%)	43 (69%)	18 (29%)	9.0 cm

In 53 of our 61 (87%) patients, the MW was more than 8 cm. Sturm and Marsh (1978) reported a MW of more than 8 cm in 60% of their patients, Fisher et al. (1981) in 75%, Barcia and Livoni (1983) in 100% of their patients. Kirsh et al. (1976) reported a widened mediastinum in 100% of their patients without giving absolute figures; Motin et al.(1980) described a MW of 10 cm or more in 92% of their 36 patients.

None of our patients with TAR showed a normal MW, nor progression of MW on successive roentgenograms. This is in agreement with the data of DeMeules et al.(1971), Fleming and Green (1974) and Ayella et al. (1977).

Akins et al. (1981) however, described several patients with a normal mediastinum on admission who developed widening of the mediastinum later. If a large hemothorax is present, it is important to drain the hemothorax in order to be able to evaluate the mediastinum and the diaphragmatic contours, as is the case when a tension pneumothorax is present.

Of the control patients, one patient had a MW less than 6 cm, 18/62 (29%) had a

MW of 6-8 cm, 25/62 (40%) of 8-10 cm, 18/62 patients (29%) more than 10 cm, 43/62 patients (69%) more than 8 cm ($p < 0.05$) as compared with the TAR group. The average MW was 9 cm (table III.17).
Comparing both groups, we can see that the average MW in TAR patients was only 0.7 cm more than the MW in the control patients. A mediastinal widening of more than 10 cm was more frequent in patients with TAR than in those without ($p < 0.05$). As other authors, we believe that widening of the mediastinum is an important sign of hemomediastinum, however because this widening is also often present on chest films of blunt chest trauma without TAR, it is a rather aspecific sign for TAR.
In combination with other changes in the contours of the mediastinum, it can be more specific.

2. *Mediastinal chest width ratio*
Seltzer et al.(1981) introduced the mediastinal chest width (M/C) ratio, to eliminate the effect of magnification in the evaluation of supine chest radiographs. They reported a 100% sensitivity and 15% specificity when the M/C ratio was 0.20 or above; 95% sensitivity and 75% specificity when M/C ratio was 0.25 or above; and a 85% specificity and 100% sensitivity when M/C ratio was 0.28 or above.
Sefczek et al.(1983) reported 10 patients with TAR; in nine a M/C ratio above 0.25 was present; in 42 negative cases, a M/C ratio of less than 0.25 was measured although the mediastinum of these patients had been suspected by clinicians to be abnormally wide.
In our patients with TAR, the M/C ratio was on the average 0.36 at the level of the aortic knob, 0.35 at the middle of the ascending aorta, 0.39 at the middle of the descending aorta. In the control patients, the M/C ratio at the level of the aortic arch was 0.34; 0.35 at the level of the middle of the ascending aorta and 0.41 at the level of the middle of the descending thoracic aorta (Table III.9,11 and Fig III.2).
In our series, M/C ratios were greater than those reported by Seltzer et al.(1981) and did not differ significantly at any level in each of the two groups ($p > 0.05$). This is contrary to the findings of Seltzer et al.(1981), however in agreement with the recent report of Marnochka et al.(1984), who state that M/C ratios are not clinically useful as they are of such insufficient sensitivity and specificity in confirming or excluding aortic rupture.

3. *Displacement of the trachea to the right*
Devation of the trachea to the right was present in 44 of the 61 (72%) patients with TAR and in the control group in 22/62 (36%),($p < 0.01$).
This sign is caused by a left sided local mediastinal hematoma. As the proximal descending aorta is located to the left in the mediastinum, it can be assumed that deviation of the trachea is more frequent in patients with TAR than in trauma patients without rupture. However, the fact that this sign was observed in 36% of

the control group makes it an aspecific sign for TAR. Especially in older patients, tracheal displacement can also be caused by elongation of the aortic arch. In the large series of patients with TAR (table II.8) in the literature, the percentage of tracheal displacement is 10-60%, in our patients series we find a rather high percentage (72%).

4. *Displacement of the left main bronchus*

This sign was present in 45 of the 61 patients with TAR (74%) as compared with 10/62 (16%) of the patients in the control group ($p < 0.01$). The downward displacement of the left main bronchus was evaluated subjectively. The displacement was often rather subtle, in a minority of the patients a more severe displacement existed. The displacement of the left main bronchus is caused by a local periaortic hematoma as in the case of tracheal displacement; most often tracheal displacement was combined with depression of the left main bronchus (n = 39). In the literature (table II.9), displacement of the left main bronchus occurs less frequently than that of the trachea. Kirsh et al.(1976) reported the sign positive in 30%, Motin et al.(1980) in 47% and Fisher et al. (1981) in 53% of their patients with TAR. A penetrated high KV chest X-ray, which was performed on our patients, plays an important role in the ability to detect depression of the left main stem bronchus and may contribute to the higher frequency of this finding in our patients.

5. *Displacement of the nasogastric tube (to the right)*

In 29 patients with TAR, it was possible to evaluate this sign; in the other patients no nasogastric tube was inserted at the time of the first chest X-rays. In 17/29 (59%) of the patients, the tube was displaced to the right as compared with 4/36 (11%) in the control group ($p < 0.01$). No displacement of the nasogastric tube to the left was seen. The positive sign was often (n = 13) combined with displacement of the trachea to the right. In four patients the nasogastric tube was displaced, while the trachea was in a normal position (Fig. III.6). In none of our positive cases, deviation of the nasogastric tube was the only radiographic abnormality. Mcllduff et al. (1977) were the first to describe the sign. Their 3 patients with TAR showed a positive sign, as was the case in the 7 patients of Tisnado et al.(1977). They found no displacement in negative cases. However, Gerlock et al.(1980), Cole et al.(1981), Fisher et al. (1981),Barcia and Livoni (1983), Sefczek et al.(1983), Woodring et al.(1984),reported a lower percentage of displacement of the nasogastric tube (50-80%) in a greater number of patients. In the reported patients without TAR, deviation of the nasogastric tube was not always absent (9-23%),(Wales et al., 1982).

They demonstrated that 3 of the 20 normal patients had deviation of the nasogastric tube, presumably as a result of aortic ectasia. The same authors reported in an autopsy study deviation of a nasogastric tube to the left in 3 of the 8 patients with TAR.

Nasogastric tube displacement seems to be a moderate sensitive indicator for aortic rupture; as an isolated finding it does not warrant immediate angiography. It is advisable to insert a nasogastric tube in blunt chest trauma patients, not only to decompress the often present gas accumulation in the stomach, but also to be able to evaluate the position of the esophagus.

6. *Loss of sharpness of aortic contours*

This sign was seen in nearly all our patients with TAR: 58/61 (95%). However, in the control group it was also frequently present: 38/62 (61%), ($p < 0.05$). The sign is caused by peri-aortic hematoma, making lung-aorta contact impossible. Loss of sharpness of aortic contours can be regarded as a silhouette sign, described by Felson (1973). Our data are in agreement with the literature where an unsharp aortic contour in TAR patients is reported in 53-100%, in controls in 39-100% (Flaherty et al., 1969; Redman, 1973; Sturm et al., 1974; Kirsh et al., 1976, Gerlock et al., 1980; Fisher et al., 1981; Barcia and Livoni, 1983; Schumacher et al., 1983; Sefczek et al., 1983; Woodring et al., 1984.)

An unsharp contour is an sensitive but nonspecific sign for TAR and conversely a sharp contour nearly always excludes an aortic rupture.

7. *Broadening of the paratracheal stripe*

This recently described sign (Woodring et al., 1982) was found in 44/53 (83%) of the patients with TAR. In the control group this sign was present in 10/34 (29%), ($p < 0.01$) of the patients. Woodring et al. (1982) discovered no arterial injury in patients with a stripe smaller than 5 mm; we had 9 aortic ruptures in patients with a stripe smaller than 5 mm. In none of our patients with aortic rupture was a broadening of the stripe an isolated finding. Together with other signs this seems a valuable and rather sensitive but nonspecific indicator of aortic injury, although a normal stripe does not exclude aortic rupture and sometimes the stripe is not visible at all.

8. *Displacement of the right paraspinal line*

The right paraspinal line was displaced in 24/47 (51%) of the patients with TAR; in the controls 4/51 (8%), ($p < 0.01$). This makes it a rather specific sign for TAR. The displacement is evoked by a posterior mediastinal hematoma. Compared with the literature, our figure is rather high: Marsh and Sturm, (1976): 16%; Peters and Gamsu, (1981): 57%, Fisher et al., (1981): 10%, (table II.12).

In the patients without TAR a displaced right paraspinal line was not reported, except by Barcia and Livoni (1983) in 14%. One of the prerequisites for being able to evaluate the right paraspinal line is a well penetrated chest roentgenogram or an X-ray of the vertebral spine. In evaluating our chest X-rays of trauma patients it was evident that a right paraspinal line was not visible at all in 20/50 patients (40%), which is in agreement with the statement of Fraser and Paré (1978) that a normal right paraspinous interface is not always visible.

9. *Displacement of the left paraspinal line*

The left paraspinal line was displaced in 38/46 (83%) of the patients with TAR; twice as high as in the control group 21/51 (41%),($p<0.01$). This is lower than in the report of Barcia and Livoni(1983): 83% and Peters and Gamsu (1981): 55%. Compared with other authors (table II.13) our figure is rather high; Sturm et al., (1979): 16%; Fisher et al., (1981): 12%. In chest trauma patients without TAR, displacement of the left paraspinal line is reported in only 25% (Redman, 1973; Fisher et al., 1981; Peters and Gamsu, 1981; Barcia and Livoni, 1983) compared with our 41%.

The left paraspinal line is more frequently displaced (38/46; 83%) than the right one (24/47; 51%); however, in the control group left sided displacement (21/51; 41%) is also more frequently found than the right sided one (4/51; 8%). It is a valuable sign for hemomediastinum, its absence however does not exclude TAR.

10. *Fracture of the first or second rib*

First or second rib fractures were found in only 5/61 (8%) of patients with TAR versus 15/62 (24%) in the control group ($p<0.01$).

Contrary to the opinion of DeMeules et al. (1971), O'Sullivan et al.(1972) and Richardson et al.(1979), first or second rib fractures seem not to be an indication for angiography when other signs indicative for hemomediastinum are absent. This is in agreement with the more recent literature (Seltzer et al., 1981; Barcia and Livoni, 1982; Woodring et al., 1982; Gundry and Kirshner, 1983; Livoni and Barcia, 1982). The present study failed to show that first or second rib fractures are more highly correlated with aortic tear than with rib fracture at any other level or with no rib fracture at all.

11. *Other rib fractures*

Rib fractures other than of the first or second were present in 24/61 (39%) in TAR patients and in 28/62 (45%) of the controls ($p>0.05$).

Rib fractures, especially in younger patients are not a constant feature as elasticity of the thoracic cage is great (Soyer et al., 1981).

12. *Left apical cap*

This sign was present in 42/61 (69%) of the patients with TAR, in the control group in 33/61 (54%) of the patients ($p>0.05$). The apical cap must be explained by the spread of the mediastinal hematoma in the extrapleural space as in only 5 of the TAR patients first or second rib fractures were present. Our data are in agreement with those of Barcia and Livoni (1983).

Contrary to the belief of Simone et al (1975) that a left apical cap is one of the earlier signs of aortic injury, we found no cases in which this cap was the only finding in TAR patients. Furthermore, in the control group the cap was visible with almost equal frequency. Other authors (Fisher et al., 1981; Peters and Gamsu, 1981; Barcia

and Livoni, 1983; Sefczek et al., 1983) held the same opinion: there is no significant difference in the occurence of apical cap in trauma patients with and without TAR.

13. *Hemothorax*

Left sided hemothorax was found in 20/60 (34%) of the patients with TAR, in control patients in 18/62 (29%), ($p > 0.05$).

Left hemothorax was present in only 3 of the 14 positive cases reviewed by Peters and Gamsu (1981). It was also seen in two of their 14 angiographically negative examinations and as such was described as a relative insensitive and nonspecific finding in TAR.

A massive hemothorax is seldom a sign of TAR, because it means that there is abundant extravasation from the aorta and the patient will (usually) not survive. If a massive hemothorax is present in patients with blunt thoracic trauma it is important to perform a pleural drainage in order to be able to evaluate the mediastinum and the position of the trachea. The same holds for a tension pneumothorax. Hemothorax probably is most often caused by rib fractures, not by the aortic rupture. Other causes for massive hemothorax in chest trauma patients can be lesions of the intercostal vessels, a ruptured bronchus and injury of the pulmonary vessels.

14. *Displaced superior vena cava*

This sign is difficult to evaluate because no objective parameters are offered in the literature. We observed a displacement of the superior vena cava in 29/58 (50%) of patients with TAR and in 19/62 (31%) of the control patients ($p < 0.05$).

15. *Opacified pulmonary window*

This sign was positive in 58/60 (97%) of our patients with TAR, as compared with 31/61 (50%) of our control patients ($p < 0.01$). Unfortunately evaluation of this sign has to be subjective, as no clear cut parameters are available for evaluation of the pulmonary window. We have relied on the sign however, because of the high sensitivity particulary when combined with other parameters in order to distinguish patients with TAR.

Other signs

In two older patients, a distinct inside displacement of intimal calcification as a sign of aortic wall changes indicating aortic rupture was present. The intimal calcification was displaced over a distance of 1.5 cm. Anterior displacement of the trachea on the lateral chest radiographs as described by Molnar and Pace (1966) could not be evaluated in our patients because in our trauma patients a lateral chest film was only infrequently made.

From our data and from the literature it is clear that clinical symptoms indicating TAR are scarce: only the coarctation syndrome and acute hoarseness were specific symptoms.

The chest X-ray in blunt chest trauma patients is essential for diagnosing TAR. Signs of mediastinal bleeding that were significantly more frequent in patients with TAR as compared with the control patients were: displacement of the trachea to the right; downward displacement of the left main bronchus; displacement of a nasogastric tube to the right; unsharp aortic contours; opacified pulmonary window; widened left and right paraspinal lines; broadened paratracheal stripe (All signs $p<0.01$). Displacement of the superior vena cava was more frequent in the TAR group too ($p<0.05$).

Mediastinal widening, both 8 cm or 10 cm was more frequent in the TAR group in comparison with the controls ($p<0.05$). Mediastinal chest width ratios were found to be relative high in both groups and were not useful in differentiating the groups ($p>0.05$)

From the statistical evaluation (Appendix B) the 'best' signs to distinguish patients with hemomediastinum without rupture from those with TAR appeared to be an opacified pulmonary window; a broadened right paratracheal stripe; a widened right paraspinal line and a displaced nasogastric tube.

In general in 4 to 6 patients with hemomediastinum angiography of the thoracic aorta has to be performed, to detect one patient with TAR. (Bodily et al., 1977; Richardson et al., 1979; our experience).

We therefore estimate that the a priori probability of an aortic rupture in a blunt chest trauma patient with signs (table III.8) of hemomediastinum is 20%. Combining this information with variables known to have a reasonably good discriminating power between both groups (chest trauma patients with and without TAR) we

Table III.18 A posteriori probability of TAR for combinations of two signs from the chest X-ray in patients with hemomediastinum[1]

Signs	*TAR patients*	*Control patients*	*A posteriori probability for TAR*
Opacified pulmonary window Widened paratracheal stripe	n = 43 (83.7%)	n = 4 (12.1%)	63% (SE[2] 11%)
Opacified pulmonary window Widened right paraspinal line	n = 24 (52.2%)	n = 3 (6%)	68.5% (SE 12.5%)
Widened paratracheal stripe Widened right paraspinal line	n = 20 (47.6%)	n = 1 (3.2%)	78.7% (SE 16.7%)
Displaced nasogastric tube Widened paratracheal stripe	n = 15 (60%)	n = 1 (5%)	73% (SE 19.4%)

[1] Probability of no rupture is 100% minus these probabilities.

[2] SE Standard Error Large SE is due to relative small number of patients.

Table III.19 A posteriori probability of TAR for combination of three signs from the chest X-ray in patients with hemomediastinum[1]

Signs	*TAR patients*	*Control patients*	*A posteriori probability for TAR*
Opacified pulmonary window Widened paratracheal stripe Widened right paraspinal line	n = 20 (48.8%)	n = 0 (0%)	100% (SE 16.8)
Opacified pulmonary window Widened paratracheal stripe Hemothorax	n = 13 (25.5%)	n = 2 (6.1%)	51.3% (SE 18.1)

[1] Probability of no rupture is 100% minus these probabilities.

can calculate - using Bayes rule - the a posteriori probability of TAR with combinations of two and three discriminating variables in our patient population (table III.18 and III.19).

A combination of more than three signs was not investigated, as numbers of patients would be too small and standard deviations too large.

With a combination of an opacified pulmonary window, a widened paratracheal stripe and a widened right paraspinal line the a posteriori probability of TAR was the highest: 100%(SE 16.8). This means that of 100 patients with blunt chest trauma, undergoing angiography and showing these three signs on the chest X-ray, 100 are expected to have an aortic rupture, but due to the large standard error, this may be considerably smaller. Unfortunately an opacified pulmonary window is a sign that can be assessed only subjectively as no objective measurements for the evaluation of the window are known. Woodring et al.(1984), making an analysis of sensitivity and specificity of radiographic signs on the chest X-ray for TAR, found a displaced nasogastric tube and a widened paratracheal stripe the best predictors for TAR (24% positive correlation). When combinations of two or three of the 'best' discriminating signs tabulated in table III.18 and 19 are absent the a posteriori probability for TAR is low: 0% (Appendix B).

Conclusion:

If one or more signs of hemomediastinum are present on the chest X-ray of a patient with blunt thoracic trauma, angiography is indicated in order to exclude the possibility of TAR. Some signs or combination of signs are more sensitive indicators for TAR compared with others; no signs have a specificity of 100%. A combination of an opacified pulmonary window, a widened paratracheal stripe and a widened right paraspinal line have the best predictive value (100% ± 16.8) for TAR in patients with hemomediastinum. Conversely, when these three signs are absent, the probability for TAR is low: 0% (SE 1.5%), (Appendix B). All other signs or

combinations have a lower predictive value (51-75%) for TAR. Because not always all signs are present or can be evaluated on the chest X-ray, we estimate that even with much experience, the angiography in only one out of two or three chest trauma patients with hemomediastinum would be positive for TAR.

III.4.4. *Angiography*

In agreement with other authors Kirsh et al. (1976); Vasko et al.(1977); Akins et al.(1981), it is our opinion that angiography should be performed in patients suspected of TAR. It is important to localise the rupture(s), as false aneurysms of the aortic arch (can) make another operative approach necessary, and ruptures can be multiple. Furthermore, injuries to the brachio-cephalic vessels can also be excluded with angiography. The most important reason however is, that the described signs on the plain chest X-ray indicate to a greater or lesser extend the possibility of TAR, but not sufficiently accurate.

Although the operative morbidity and mortality for thoracotomy is low, unnecessary thoracotomy should be avoided.

Angiography was performed in 52/57 patients, immediately after suspicion for TAR was raised on basis of the chest X-ray. In two patients, angiography was not performed because aortic rupture was not suspected, three patients died before angiography could be performed. We were able to review all angiograms. In 17 patients angiography was performed 1-3 hours after trauma; in 12 patients 3-6 hours after trauma; between 6 and 24 hours in 7 patients; in 10 patients later than 24 hours after trauma; in 6 patients the exact time of angiography was not known.

In most patients (n = 49) a femoral approach was used. In one patient a brachial entry was used because of the absence of the femoral pulses. In five patients it was not possible to pass the traumatised segment of the aorta; in two of these patients the aorta was completely transected, in two patients a dissection was shown. Contrast injection with a pressure device and the catheter in the false aneurysm seems us to carry risks for complete rupture although in some cases of the present series it was done without complications. It seems more advisable, when the catheter cannot pass the injured segment of the aorta or is snapped in the false aneurysm, to use an additional brachial or axillary approach. A complementary angiography by the brachial route was made in two patients. 47 angiograms were performed in the University Hospitals, five patients were referred with an angiogram that was performed elsewhere. In one case, a DSA investigation was performed in a community hospital, and while interpreted as negative, angiography was positive for TAR. We have the opinion that a successful intravenous DSA investigation is possible only when the patient can be cooperative, in particular with regard to movement and of breathholding. This will often be impossible with these multi-trauma patients in respiratory distress. In the majority of patients a 'classic' angiography has to be the

preferred method. An alternative approach can be intubation and mechanical ventilation: in those patients a DSA investigation can be performed during apnea. It is not clear from our data nor from the literature whether angiography should be performed in the referring clinic when there is suspicion for TAR or in the hospital in which the (potential) TAR patient would be operated.

Four false aneurysms were localised in the aortic arch, 48 at the isthmus, four in the descending thoracic aorta and one at the level of the diaphragmatic hiatus. Multiple ruptures were present in one patient: one at the aortic isthmus and one in the descending thoracic aorta. No ruptures of the ascending aorta were seen. These findings do not differ from other reported series: Symbas (1972); Thevenet (1975); Bodily et al.(1977); Turney et al.(1977); Barcia and Livoni (1982).

An aneurysm seated on the outside contour of the aorta was seen in 9 patients, a localisation on the inner curvature of the aorta in 15 patients. Circumferential tears were visible in 18 patients. In the other patients, exact localisation was not possible.

In three patients, lesions of the brachio-cephalic vessels were seen in combination with TAR: two had a false aneurysm of the left subclavian artery; one patient a dissection of the left carotid artery.

In general, the time required for angiography was 30-45 minutes; in the University Hospital of Groningen angiography was performed in the emergency room with C arm equipment, which required less than 30 minutes; in the trauma unit in Nijmegen, patients had to be transported to another room for angiography, which required more time. It is obvious that angiography without transport of the patients and with a C arm equipment is to be preferred.

In one patient without femoral pulses the brachial puncture was difficult and took even one hour. After angiography when the patient was lifted from the table, he died suddenly. A difficult puncture can be a reason for time delay and one should not hesitate to ask for an arteriotomy. Angiography in these situations should be carried out only by experienced angiographers.

In 18 patients, angiography of the abdominal organs was performed in the same session. Careful clinical judgement, whether to perform abdominal angiography or not, is necessary because abdominal angiography can result in unnecessary delay in case of frank abdominal bleeding, for which immediate operative treatment is the only correct approach. On the other hand, especially when systemic heparinisation is planned it seems justified to exclude damage to liver, kidney and spleen, which can be done by ultrasonography or CT scanning.

Complications

No complications were recorded except in one patient in whom the catheter entered the false aneurysm and contrast extravasation in the mediastinum was seen on test injection. No angiography was performed and during celiotomy the patient died from massive hemorrhage of the liver.

We agree with the opinion of most authors that angiography should be performed immediately when a TAR is suspected. It makes the diagnosis sure and the false aneurysm can be localised better, which is important for the surgical approach. Angiography can be performed quick and without risks for the patient. No serious complications due to the angiographic investigation were encountered. A femoral approach is the most convenient both for the patient and the physician, except when femoral pulses are absent. Abdominal angiography can most often be replaced today by echography and/or computer tomography. The future role of DSA and CT in diagnosing TAR has to be awaited.

Chapter IV

CONCOMITANT TRAUMATIC FINDINGS IN PATIENTS WITH TAR AND IN CONTROL PATIENTS

IV.1 **Introduction**

The management of an exsanguinating patient with multiple injuries is always a clinical challenge. Not only is the correct diagnosis important, but the classification of the injuries according to their severity and determining the temporal sequence of surgical intervention is essential for a favourable outcome. In the majority of TAR patients concomitant injuries of the thorax and/or other organs are present; often these are lethal injuries, and the patients are unsalvageable (Greendyke, 1956; Bodily et al., 1977). These concomitant injuries can be the reason for substantial operative or pre- and postoperative morbidity and mortality. One third of the patients of Bodily et al. (1977) had concomitant lethal injury and were unsalvageable.

We will discuss separately concomitant traumata to the chest and other organs.

Table IV.1 Concomitant thoracic injuries in patients with TAR

Year	*Author*	*Nr. patients*	*Fractured ribs or sternum*	*Fractured vertebrae*	*Hemo- (pneumo) thorax*	*Diaphragm rupture*	*Lung contusion*	*Heart damage*
1947	Strassmann*	72	58	4				
1958	Parmley*	275		34				104
1962	Zeldenrust and Aarts*	88		8				22
1966	Greendyke*	42	30	14			10	17
1971	DeMeules et al.	12	8		5		3	
1971	O'Sullivan et al.	12	6	2	3	1		1
1974	Fleming and Green	10	4		5			
1976	Kirsh et al.	43	28					
1977	Ayella et al.	36	5		1			
1977	Bodily et al.	39	8		7	4	5	12
1977	Pickart et al.	22	6		7	1	1	
1977	Turney et al.	31	34		21		18	2
1977	Vasko et al.	19	9					
1978	Lacquet et al.	9	3		2		3	
1979	Richardson et al.	36	12			3		
1981	Akins et al.	44	28		4	2	25	

* Autopsy series.

IV.2 **Literature**

IV.2.1 *Frequency of concomitant thoracic injuries in patients with TAR*

Table IV.1. provides literature data regarding the frequency of concomitant traumatic thoracic injury in patients with TAR.

As can be seen from table IV.1, cardiac injury, especially in the autopsy series, was rather frequent. The other most common injuries were fractures of the thoracic cage or spine, hemo-or pneumothorax, diaphragmatic rupture and lung contusion.
Another presentation of concomitant thoracic injuries and aortic injury is offered by Heberer (1971). Out of 389 lethal blunt thoracic injuries there were 160 aortic injuries of which 88 were lethal, 72 aortic ruptures were concomitant to other lethal injuries.
In the autopsy series of Zeldenrust and Aarts (1962) the lethal aortic rupture was the only severe lesion in 30 of the 88 patients with TAR.

Table IV.2 Concomitant extra-thoracic injuries in patients with TAR

Year	*Author*	*Nr. Patients*	*Abdominal organs*[1]	*Extra-thoracic fractures*	*Central nervous system injury*
1958	Parmley et al[2].	275	?	27	18
1962	Zeldenrust and Aarts[2]	88	8	19	
1966	Greendyke[2]	42	40	4	
1971	DeMeules et al.	12	5	5	4
1972	O'Sullivan et al.	12	3	4	
1972	Plume and de Weese	15	?	1	9
1973	Symbas		28		
1974	Fleming and Green	10	10	3	
1976	Kirsh et al.	43	12		24
1976	Turney et al.	31	15	16	6
1977	Aycila et al.	36	23	30	6
1977	Bodily et al.	37	18	8	6
1977	Pickard et al.	22	11	14	4
1977	Vasko et al.	19	4	15	4
1978	Lacquet et al.	9	3	6	2
1979	Richardson et al.	36	3	26	6
1981	Akins et al.	44	8	27	18

[1] Abdominal organs: ruptured spleen, liver, pancreas.
[2] Autopsy series

IV.2.2 *Frequency of extra thoracic injuries in patients with TAR*

Table IV.2 shows literature data regarding the frequency of concomitant traumatic injuries to other organs in patients with TAR.
As can be seen from table IV.2, injuries to the abdominal organs (e.g. rupture of the liver, the spleen and the intestine), extra-thoracic fractures and CNS (Central Nervous System) trauma were rather frequent in patients with TAR. The most frequent causes of complications and death from concomitant trauma were

- central nervous system trauma (Kirsh et al., 1976; Ayella et al., 1977; Pickard et al., 1977; Richardson et al., 1979)
- spleen and liver rupture (Bodily et al., 1977)
- extensive pulmonary contusion and hypoxia (Kirsh et al., 1976).

On the other hand, exsanguination from the aortic rupture can occur when severe intra-abdominal lesions are operated upon first (Kirsh et al., 1976).
Another indirect cause of death due to concomitant trauma is excessive hemorrhage as a result of the heparinisation necessary in some bypass procedures (Vasko et al., 1977). It seems therefore important in each individual case to evaluate which injury has to be repaired first and what kind of surgical technique and spinal cord protection method is most appropriate.

IV.3 **Own patients (Nijmegen and Groningen)**

Concomitant traumatic injuries were present frequently in our 63 patients with TAR. They were an important cause of operative or pre- and postoperative morbidity and mortality. These concomitant injuries were located in the chest in 26/60 (43%) of the patients and in other organs in 50/60 (83%) of the patients. In 34/60 (57%) of the patients, there were no concomitant injuries of the thoracic organs and in 10/60 (17%) of the patients no concomitant injuries of other parts of the body. Four patients had no concomitant injuries at all. In chest trauma patients without TAR, we found concomitant injuries of the chest in 30/62 (48%) of the patients ($p > 0.05$ compared with TAR patients), of other organs in 30/41 (73%) of the patients. No concomitant injuries of the chest were present in 32/62 (52%) of these patients and non in other organs in 11/41 (27%) of the patients. We will discuss these concomitant injuries in more detail.

IV.3.1 *Concomitant thoracic injuries in our patients with and without TAR*

Traumatic injury of thoracic organs apart from the aorta was often present in our patients with trauma of the chest, particularly of the lungs, heart, thoracic spine, diaphragm and pleura (Table IV.3).

Table IV.3 Concomitant injuries to thoracic organs in our patients with and without TAR[1]

	TAR patients		*Control patients*		*P*
Lung contusion	22/60	36%	30/61	49%	>0.05
Diaphragmatic rupture	6/59	12%	0/41	0%	<0.05
Pneumothorax	6/60	10%	16/60	27%	<0.05
Sternal fracture[2]	5/34	15%	6/41	14%	>0.05
Fractured vertebrae	6/58	10%	7/41	17%	>0.05
Heart injury[3]	11/35	30%	1/28	3%	<0.05
No concomitant thoracic injuries	34/60	56%	32/62	52%	>0.05

[1] Data from the chest X-ray or operative findings.
[2] No lateral chest X-ray or sternum X-ray obtained in most patients.
[3] Contusion, rupture, pericardial blood.

It is interesting to note that in 34/60 (57%) no concomitant injuries of the chest organs, with the exception of rib fractures, were present in the patients with TAR. Comparing both groups, there were slightly fewer concomitant injuries of thoracic organs in patients with TAR than in the control patients, except for diaphragmatic rupture, heart injury and pneumothorax.

IV.3.2 *Concomitant extra-thoracic injuries in our patients with and without TAR (Table IV.4)*

Table IV.4 Concomitant injuries to extra-thoracic organs in our patients with and without TAR

	TAR patients		*Control patients*		*P*
Splenic rupture	16/57	28%	4/40	10%	<0.05
Kidney rupture	3/57	6%	2/40	5%	>0.05
Liver rupture	10/57	18%	1/39	2%	<0.05
Multiple extremity fractures	38/59	64%	20/40	50%	<0.05
Intestine perforations	6/56	11%	1/39	2%	<0.05
Epi/subdural hematoma	4/58	7%	4/39	10%	>0.05

As can be seen from table IV.4, it is clear that concomitant injuries to extra-thoracic organs were more severe in patients with TAR as compared to the control patients.

This was especially true for rupture of the spleen and liver. It does not seem unlikely that this is a reflection of the greater severity of the impact at the time of the trauma in patients with TAR as compared with the controls. Death related to concomitant trauma will be discussed in chapter VI.3.2.

IV.4. **Discussion**

The high impact often present in deceleration trauma, frequently results in multiple organ injury. Apart from TAR, concomitant injuries threaten the patient's life. Frequency of concomitant injuries to thoracic organs did not differ in patients with TAR when compared with the control patients, except for diaphragmatic rupture and heart injury, which were more frequent in TAR patients. Another interesting finding was that in 57% of patients with TAR, no concomitant injuries to the chest, except for rib fractures, were present. The more frequent presence of abdominal lesions in TAR patients probably reflects the greater severity of the impact to which patients with TAR were subjected.

Our findings do not essentialy differ from other clinical series. (DeMeules et al., 1971; O'Sullivan et al., 1972; Pickard et al., 1972; Fleming and Green, 1974; Richardson et al., 1975; Kirsh et al., 1976; Ayella et al., 1977; Bodily et al., 1977; Turney et al., 1977; Vasko et al., 1977; Plume and DeWeese 1979; Akins et al., 1981), although comparative data of TAR and control patients are not given. Contrary to the opinion of Rhys-Davies et al. (1970) who stated that the combination of TAR with diaphragmatic rupture is rare, we observed this combination 5 times. Epi- and subdural hematoma were present with equal frequency in both groups. See also the discussion in chapter V.

Chapter V

SURGICAL THERAPY

V.1. **Introduction**

In 1902 Tuffier unsuccessfully attempted removal of a thoracic aortic aneurysm by surgery. Weisel et al.(1951) have been credited with the first planned operation on a chronic traumatic aneurysm of the aorta, in which cellophane wrapping was employed. Bahnson (1953) performed the first resection of an aneurysm, using lateral incision of the aneurysm. The first successful excision of a chronic traumatic thoracic aneurysm was reported by Stranahan et al.(1953), while the first repair for acute rupture of the aorta was performed by Klassen and reported by Passaro and Pace (1959). The experience that the untreated aortic rupture follows an unpredictable course, makes many authors feel that aside from rapidly progressive cerebral injuries such as epidural hemorrhage or massive intra-abdominal bleeding, traumatic rupture of the aorta deserves the highest priority and its repair should be carried out urgently. Direct repair of the rupture requires crossclamping of the aorta and interruption of the distal flow, which means that organs supplied by the distal aorta can experience an inadequate blood supply. The kidney and particularly the spinal cord are especially vulnerable to even brief periods of ischemia (Crafoord, 1947; Adams and van Geertruyden, 1956). This results from the highly variable course and origin of radicular arteries, which may result in the inability of collateral function by the anterior spinal artery. Also, when crossclamping the aorta, hypertension in the cerebral vessels should be prevented as well as acute left heart failure. Stranahan et al.(1952) stated that the risk for postoperative paraplegia is dependent on 4 factors:

1. time of crossclamping
2. level of crossclamping
3. number of intercostal and collateral vessels damaged
4. level of metabolic rate below the obstruction.

V.2. **Literature**

V.2.1. *Surgical technique*

Exposure of the aorta is usually obtained by a posterolateral thoracotomy through the left fourth intercostal space. A median sternotomy is used in patients with aneurysms of the ascending aorta or innominate artery. If a shunt is used, cannula-

tions are performed after systemic heparinisation, whereafter the proximal and distal parts of the descending aorta are clamped. The extensive mediastinal hematoma, the surrounding reaction, the retraction of the two ends of the aorta and the friability and/or dissection of the injured aortic wall, usually necessitates the use of prosthetic material to bridge the gap in the aorta after resection of the injured tissue (Braitman and Dart, 1976; Kirsh et al., 1976; Pickard et al., 1977; Vasko et al., 1977; Plume and DeWeese, 1979; Richardson et al., 1979). Although the use of homograft replacement was advised in the early days of aortic rupture surgery (Spencer et al., 1961), the availability of good prosthetic grafts has made the use of homografts less attractive; now they are recommended only if a prosthesis has become infected (Pate et al., 1968; Pickard et al., 1977).

Some authors favor the use of direct anastomosis when possible (Groves, 1961; Stoney et al., 1964; Brettschneider and McClenathan, 1965; Bromley et al., 1965; Alley et al., 1966; Beall et al., 1969; Fontan et al., 1972; McCough and Hughes, 1973).

Advantages of primary repair listed by McCough and Hughes (1973) are: shortened operation time (only one suture line); less blood loss as there is no prosthesis leakage; no risk of infection of prosthesis and no uncertainty regarding the long term results in contrast to the unknown fate of a vascular prosthesis. However, because of the earlier mentioned circumstances, most surgeons use a graft as the preferred method for repair of aortic rupture (Lacquet et al., 1975; Braitman and Dart, 1976; Kirsh et al., 1976; Ayella et al., 1977; Pickard et al., 1977; Vasko et al., 1977; Plume and DeWeese, 1979; Richardson et al., 1979).

V.2.2. *Methods used to prevent distal ischemia*

Despite unanimity on the indication for immediate operation of the aortic rupture, the best technique to protect the distal circulation remains controversial (DeBakey and Cooley, 1953; Crawford et al., 1970, 1973; Appelbaum et al., 1975; Kirsh et al., 1976; Akins et al., 1981). Operations for the rare traumatic aneurysm of the ascending aorta require total heart -lung bypass and will not be discussed.DeBakey and Cooley (1954) used systemic hypothermia during resection and grafting of a chronic traumatic aneurysm with recovery in spite of 60 minutes of aortic occlusion. This method is no longer used because of cardiac, pulmonary and hematologic complications (Cooley et al., 1957). Temporary local aortic bypass shunts like homografts (Adams and van Geertruyden, 1956) or prosthetic tubes have been sutured from the aortic arch or subclavian arteries to the distal descending aorta. Artificial materials such as Tygon and Dacron, glass and heterologous artery have been used as shunts (Stranahan et al., 1955; Symbas, 1972), with the advantage that no systemic anticoagulation is needed (Cooley et al., 1957). The bypass provides good distal protection of the abdominal viscera and the spinal cord.A disadvantage of these local

bypass techniques in case of severe mediastinal bleeding, is the risk of complications as local bleeding at the site of the anastomosis (Symbas, 1972; Cukingnan et al., 1974). Partial left heart bypass (left atrium to femoral artery) with an interposed extracorporal pump as described simultaneously by Gerbode et al. (1957) and Cooley et al. (1957), has become a common method for prevention fo distal ischemia (Keen, 1969; Mulder and Grollman, 1969; Lacquet et al., 1972). The use of an oxygenator has been advocated by DeMeules et al. (1971) to compensate for the posttraumatic hypoxia caused by pulmonary contusion. Because occasionally problems have risen with this technique due to a small left atrium, cardiac trauma or excessive mediastinal bleeding, modifications have been developed: femoral vein-femoral artery bypass (Bloodwell et al., 1968; Beall et al., 1969; Aronstam et al., 1971; Symbas, 1972; Dart and Braitman, 1976; Richardson et al., 1979). This technique requires a pump oxygenator. Left axillary to left femoral bypass is advocated by Pate et al. (1968). With these bypass techniques systemic heparinisation is necessary.

From the literature (a.o.Vasko et al., 1977) it appears that the use of systemic heparinisation is associated with significantly higher morbidity and mortality rates, mainly because heparinisation may increase intra-abdominal or intra-cerebral bleeding. It is possible to use arterial cannulae composed of plastic as external shunts (Kirsh et al., 1970, 1976). Tridodecyl methyl ammonium chloride (TDMAC)-heparin coated shunt according to Gott is a commercially available external shunt. These external shunts can be used without heparinisation (Connors et al., 1975). Aortic-aortic bypass without pump or heparinisation was used by Crawford and Rubio (1973), Gazzinga et al. (1975), Ayella et al. (1977) and Turney et al. (1977).

Crawford and Rubio (1973) challenged the view that a bypass is needed to prevent distal ischemia: they demonstrated from the literature and from their own observations that the incidence of paraplegia and other complications was not higher when the upper descending aorta was clamped without a shunt than when a shunt was used, as long as the time of clamping did not exceed 45 minutes and hypotension did not develop.

The same conclusion was reached by Brewer et al. (1972) relative to the repair of coarctations. In their opinion, systemic blood pressure is the single most important factor controlling blood flow to the vessels of the spinal cord. Appelbaum et al. (1975) found no cases of paraplegia in 8 patients operated without a shunt; a shorter operation time was an additional gain. Vasko et al. (1977) found no cases of paraplegia in 6 patients operated without shunt, versus 1 in 23 with aorta-aorta shunt. Proximal aortic pressures were kept below 140 mm Hg. by administration of nitroprusside during aortic occlusion. Vasko et al. stated: 'this technique was attended by a decrease in mortality, paraplegia, blood loss, operating time and general morbidity'. Turney et al. (1976) and Pickard et al. (1977), using the same technique, held the same opinion.

In The Netherlands, Skotnicki et al.(1982) and Buskens et al. (1984) applied the same technique. Simple crossclamping is very useful in the presence of severe concomitant abdominal or cerebral injuries as systemic heparinisation can be avoided and the risk of bleeding is diminished.
In summarising the literature, it seems clear that the trend has shifted away from the left heart bypass, because of the longer operation time and the risk of complications from heparinisation towards either simple crossclamping the aorta or the use of a shunt not requiring heparinisation.

V.2.3. *Other operations*

If serious intra-abdominal or intra-cerebral bleeding is present, operations to stop the bleeding should be performed first, especially if a bypass technique with heparinisation is to be used for the aortic repair (Pickard et al., 1977). This is however not without risk: of six patients reported by Kirsh et al.(1976) undergoing at first repair for abdominal injury, five died of exsanguinating hemorrhage from the aortic rupture, while none of six patiens who underwent celiotomy immediately after repair of aortic rupture died. In those circumstances in which the repair of the aortic rupture must be delayed because another operation should be performed first, it is important to lower the systolic blood pressure below 120 mmHg with antihypertensive agents (Aronstam et al., 1970; Kirsh et al., 1976; Akins et al. ,1981). Ayella et al.(1977) stated:' our treatment priorities are based on results of a miniature laparotomy. If bright blood is seen as soon as the peritoneum is opened the abdomen takes precedence. If peritoneal lavage is needed to detect the abdominal hemorrhage, the aorta is repaired first.' In general, indications for immediate celiotomy are an expanding abdomen, hemodynamic instability, a positive peritoneal lavage, signs and symptoms of peritonitis, or the finding of free air under the diaphragm.

V.2.4. *Complications*

One of the most threatening complications in surgery of aortic rupture - aside from the local complications of bleeding, recurrent or phrenic nerve injury and post-pericardiotomy syndrome - is the postoperative paraplegia.Earlier in this chapter, we discussed the mechanisms of the postoperative paraplegia and methods of prevention. Table V.I and V.2 show the incidence of paraplegia in patients operated for aortic rupture with and without a shunt.
It is obvious from table V.l, that postoperative paraplegia remains a problem, spite the use of bypass techniques which carry risks by themselves and certainly add to the extent and time of the operation.
Another complication described is esophageal necrosis due to compression of the

Table V.I Paraplegia in patients with TAR operated with a shunt

Year	*Author*	*Type shunt*	*Nr. patients*	*Paraplegia*
1958	De Bakey et al.*	LHB[1]	36	–
1964	Jahnke et al.	LHB	6	0%
1968	Bloodwell et al.*	LHB	17	12%
1968	Neville et al.*	LHB	19	0%
1969	Beall et al.	LHB	5	20%
1970	Kahn et al.	External	26	4%
1972	O'Sullivan et al.	LHB	12	8%
1973	Crawford and Rubio*	Various types	38	8%
1974	Fleming and Green	Various types	43	0%
1975	Appelbaum et al.	Various types	11	8%
1976	Kirsh et al.	LHB	10	?
		External	27	0%
1977	Pickard et al.	Various types	14	7%
1977	Vasko et al.	LHB	12	0%
1978	Lacquet et al.	LHB	9	0%
1981	Akins et al.	LHB	23	0%
		External	12	8%

[1] LHB left heart bypass

* Various types of aneurysm of the descending aorta.

Table V.2 Paraplegia in patients with TAR operated without a shunt

Year	*Author*	*Nr. patients*	*Paraplegia*
1973	Crawford and Rubio*	45	2.2%
1975	Appelbaum et al.	7	0%
1976	Turney et al.	6	0%
1977	Vasko et al.	5	0%

* All kinds of aneurysms

esophageal wall by the hematoma or injury to the arterial blood supply of the esophagus (McBurney and Vaughan, 1961; Fleischaker et al., 1964; Lacombe et al., 1971; Schumacher et al., 1983). A further complication that can occur is bleeding, intra-abdominal or cerebral, resulting from heparinisation when a shunt is used. Thoracic and non-thoracic postoperative complications as a consequence of multi-organ injury and failure can occur and influence prognosis. This will be discussed in the chapter VI.

Wheat et al. (1965) presented a new approach for control of acute aortic dissections. Different pharmacological agents were used to lower the blood pressure, thus lowering the shearing jet effect of the pulse. Necessary corrective surgery was postponed until the acute phase of the illness receded to an elective time when tissues had become more amenable to surgical repair. Aronstam et al. (1970) used this technique in 4 patients with TAR without losing one patient. The systolic tension was kept between 90 and 100 mmHg. Akins et al. (1981) treated 19 patients with antihypertensive drugs in whom for various reasons an operation for TAR was delayed. The period of elective delay varied between 2-79 days. Fourteen patients were operated upon: 5 patients had concomitant injuries with such severe sequelae that operative intervention was delayed indefinitely.They state: 'it should be emphasized that the principal reason for elective delay in the early 1970's was the fear of the effect of systemic heparinisation on the other injuries. With our current preference for the use of the TDMAC shunt, the risk of extensive bleeding with heparinisation has been eliminated. Our present indications for elective delay include evaluation and stabilisation of extensive CNS trauma,extensive body surface burns that have a high risk of infection, large contaminated open wounds with high risk of infection, established sepsis, and severe respiratory insufficiency'.

V.3. **Own patients (Nijmegen - Groningen)**

Of 57 patients with TAR, 49 came to some kind(s) of operation(s). Of these 49 patients, 46 underwent a thoracotomy for aortic rupture, 3 patients had a celiotomy only (table V.3). 40 patients were operated with success for their TAR.

Table V.3 Surgery in 57 patients with TAR

	Nr. patients
Thoracotomy for TAR	46
Death before operation	8
Death during celiotomy, before TAR repair	2
Not operated for thoracic rupture*	1

* Celiotomy only

The time elapsed between the arrival at the emergency room and the first operation is shown in table V.4.

Table V.4 Time between admission and first operation in 49 patients with TAR

Time	*Nr. patients*
1-3 Hours	2
3-6 Hours	14
6-24 Hours	17
24 Hours	9
Unknown	7

V.3.1. *Surgical technique*

As soon as the diagnosis of aortic rupture was established by angiography, the patients were brought to the operating theatre. No operations for TAR were performed before angiography had confirmed the aortic rupture. All patients were operated upon by one of the staff members of the thoracic and cardiovascular departments of the University Hospitals of Nijmegen or Groningen; 46 patients were operated upon for TAR. A left lateral thoracotomy through the 4th intercostal space was performed in all instances. Identification of the phrenic, vagus, and left recurrent laryngeal nerves was attempted in all cases. After proximal and distal crossclamping of the aorta, proximal clamp generally between the left carotid and subclavian artery and another clamp on the subclavian artery, the distal clamp past the aortic isthmus, (guided by the pre-operative angiogram) with (n = 35) or without (n = 11) a shunt, the mediastinal pleura and the adventitia of the descending aorta were opened, exposing the rupture.
Fourty two of fourty six patients survived the operation for TAR (Nijmegen n = 25; Groningen n = 17). In Nijmegen a tubular prosthetic graft (woven dacron) was inserted in 23/25 patients for aortic repair (see Fig. V1. a and b). One patient with a lesion between the innominate artery and the left carotid artery was treated with a patch; another patient with a rupture in the descending thoracic aorta was treated by suturing the defect. One patient suspected by angiography of having an intimal lesion was not operated upon. In the majority (13/17) of patients in Groningen, a prosthetic graft was used for continuity repair. In the remaining 4 patients resection of the lacerated edges and end-to-end anastomosis was performed (4/17).
The length of the graft varied between 2 and 10 cm.

V.3.2. *Surgical findings*

Mediastinal and/or pleural hematoma were found in most cases. Radiological findings were confirmed in all cases except one. This was the patient with the suspected

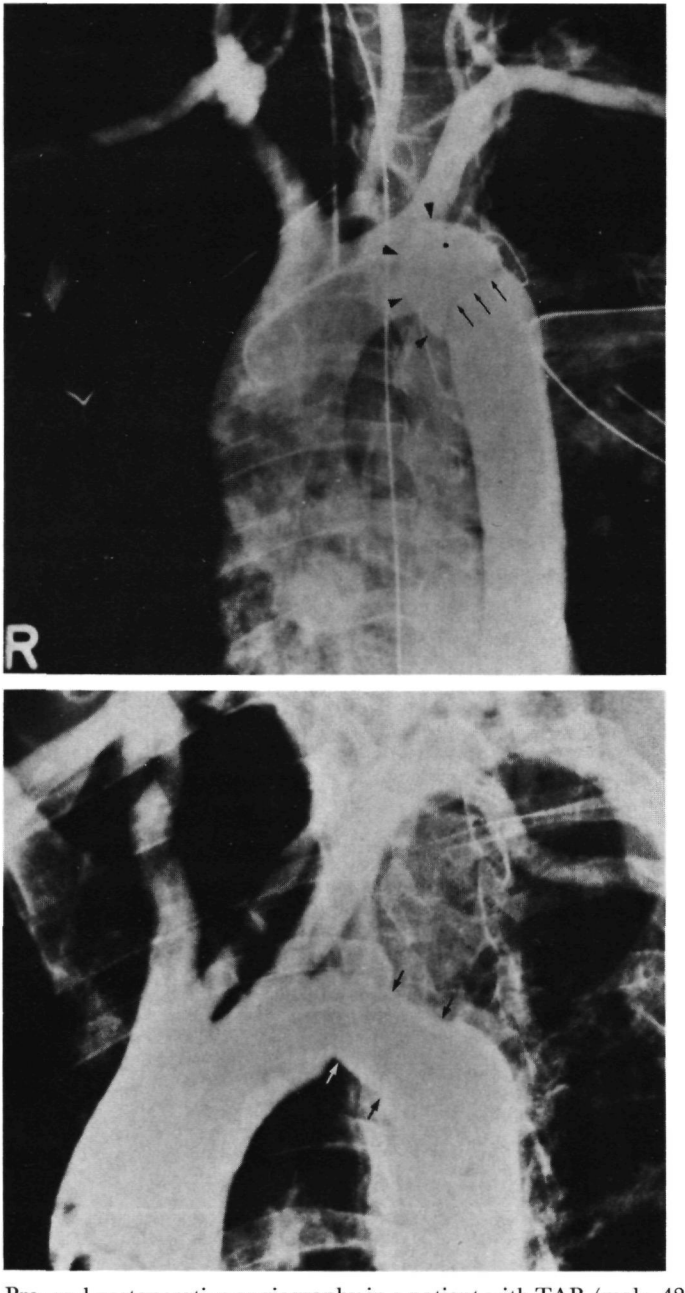


Figure V.1. Pre- and postoperative angiography in a patient with TAR (male, 42 years, car passenger, uneventful recovery). a. Preoperative angiography: typical false aneurysm at the isthmus with circumferential tear. Note intimal flap (small arrows) and the false aneurysm (triangles).
b. Postoperative angiography: at the level of the rupture a graft is interposed. Slight narrowing of the lumen at the level of the graft (arrows).

intimal tear who was not operated upon. Surgical findings with regard to the rupture are shown in table V.5.

Table V.5 Surgical findings in 46 patients with TAR

False aneurysm without transection	34
False aneurysm with transection	11
Intimal rupture	–
Multiple ruptures	(1)
Other	1

( ) Double counting.

Table V.6 The circumferential extent of the tear in 51 patients*

Circumferential Extent	*Nr. patients*
1/4	1
1/2	8
3/4	16
Complete rupture	26

* 46 at operation, 5 at autopsy.

The extent of the tear at the site of the rupture is shown in table V.6.
In five patients, separation and retraction, varying from 2 to 8 cm, of the media and intima was found during the operation.Surgical findings were in agreement with angiography in all cases except in the patient with the suspected intimal tear who was not operated upon. A complete transection of the aortic wall could be predicted by angiography only when it was not possible to pass the traumatised segment of the aorta from below (n = 5). Of the four patients where the rupture was located in the aortic arch, one patient died pre-operatively, one (intimal tear?) was not operated upon, because the surgeon was not convinced that a lesion existed in this patient, one peroperatively and the fourth patient was treated with a patch on the aortic arch between the innominate artery and the left carotid artery.

V.3.3. *Methods used to prevent distal ischemia*

During the period under investigation, various methods were used to prevent distal ischemia and left ventricular and cerebral hypertension: 1) left heart bypass (LHB); 2) TDMAC or Gott shunt; 3) simple crossclamping of the aorta under pharmacologic vasodilatation (see table V.7).

Table V.7 Methods used to prevent distal ischemia in 46 patients with TAR

Left heart bypass	30
TDMAC shunt	5
Simple crossclamping and pharmacologic vasodilatation	11

Left heart bypass (left atrium to left femoral artery connection) was used with a pump without an oxygenator. Systemic heparinisation was used in all cases of LHB. Because heparinisation was required and because hemorrhage existed, celiotomy was performed as the first operation in 17 patients. The time of the bypass varied between 30 and 90 minutes.
In the TDMAC shunt group, a connection was made between the ascending aorta and the descending aorta, below the level of the distal occlusion. No pump or heparinisation was used. To prevent heart strain and hypertension in the proximal part of the body and to achieve adequate perfusion on the distal part of the body with the crossclamping technique, a nitroprusside infusion was installed and the systolic tension was kept between 80 and 120 mmHg.

V.3.4. *Other operations and their sequence*

Due to concomitant injury, other operations on patients with TAR had to be performed frequently, especially in the presence of cerebral trauma with sub- or epidural hematoma or when obvious intra-abdominal bleeding existed, these organs were operated upon first. Another reason for performing a celiotomy first was the fear of bleeding from the injured abdominal organs due to systemic heparinisation. In one patient in whom an aortic rupture was detected after a few days had elapsed, an operation for the extremity fractures was performed first. In many cases abdominal and extremity operations were performed after aortic repair (table V.8 and V.9).

Table V.8 Sequence of operations in 49 patients with TAR

First operation	*Nr. patients*
Thoracic aorta	28
Abdominal organs	17*
Cerebral organs	3
Extremities	1

* LHB shunt 11

Table V.9 Kind of operations performed on 49 patients with TAR

Kind of operation	*Nr. patients*
Thoracotomy only	17
Laparotomy only	3
Thoracotomy + laparotomy	17
Thoracotomy + extremities	4
Thoracotomy + laparotomy + extremities	6
Thoracotomy + laparotomy + extremities + cerebral	2

V.3.5. *Complications*

All patients were treated postoperatively in the intensive care unit (head: Dr. J. Gimbrère, Nijmegen; head Dr. D.R. Miranda, Groningen).
Various periods of mechanical ventilation to support oxygenation were needed in almost all patients. Postoperative complications of the thorax and other organs are shown in table V.10 and V.11.
There were 6 deaths during the operations. One patient died due to rupture of a non-recognised TAR, during a diaphragmatic repair. In two patients, ruptures extended to the aortic arch or ascending aorta and it was impossible to crossclamp

Table V.10 Postoperative thoracic complications in 46 patients operated for TAR

Complications	*Nr. patients*
Adult respiratory distress syndrome (ARDS)	3
Pulmonary embolism	2
Other*	13

* E.g., hemo-pneumothorax, mediastinal bleeding.

Table V.11 Postoperative non-thoracic complications in 46 patients operated upon for TAR

Complications	*Nr. patients*
Abdominal complications	5
Cerebral complications	6
Paraplegia	4
Anuria	3
Sepsis	4
Various*	7

* Various: Coagulopathia, febris, bleeding from tracheostomy, instability of lumbar spine.

the aorta without interruption of flow to the cerebral vessels.These patients could not be salvaged. Two patients were operated upon in a desperate situation: the blood pressure was not recordable and the ECG showed ventricular fibrillation. The aneurysms were ruptured through the adventitia and the mediastinal pleura in the pleural space and the patients could not be resuscitated. One patient died during celiotomy from massive bleeding from liver and spleen injury. Five of the six peroperative deaths were due to the aortic rupture.

Two postoperative deaths, directly related to the LHB procedure and systemic heparinisation, were recorded. One patient developed massive intra-abdominal bleeding after termination of the repair of the aorta. At laparotomy 10 to 15 litres of blood were found in the abdomen, caused by massive bleeding from pelvic veins. Bleeding could not be stopped and the patient died. The other patient developed postoperatively untreatable clotting disturbances and died.

Postoperative paraplegia was noted in four patients, one in the LHB group three in the cross-clamping group. These patients are described in detail.

Patient B. ♀, born 1916

On 17.07.83 the patient was involved in an automobile accident and admitted elsewhere. She was referred on 18.07.83 with suspected hemopericardium. Angiography showed an aortic rupture at the isthmus. During the operation, the aorta was crossclamped for 50 minutes, without the use of a shunt and during the procedure the sytolic tension was not below 70 mmHg. A dacron graft was inserted; three intercostal arteries had to be ligated. Postoperative areflexia below the level of Th 7 occurred with poor prognosis.

Patient v.d. Z. ♂, born 1946

This patient was involved in an automobile accident on 24.08.83 and was referred the same day from another hospital. Angiography showed a rupture at the isthmus. During the operation, the aorta was crossclamped for 44 minutes without the use of a shunt and a graft was inserted. No intercostal arteries were ligated and there were no periods of hypotension. Postoperative paraplegia was noted with gradual although slow improvement.

Patient v.d. V. ♂, born 1927

This patient was operated upon on 10.02.76 for TAR after a car accident the same day. Angiography and surgery showed a rupture at the isthmus with complete transection of the aorta. A graft was inserted after crossclamping the aorta and the use of a LHB shunt. There were some periods of peroperative hypotension due to concomitant cardiac injury. Postoperative paraplegia of both legs was present. The patient died on the 41st postoperative day due to sepsis.

Patient Be, ♀ born 1959
This patient was operated upon for TAR on 22.03.82 after an automobile accident the same day. Angiography showed a rupture at the isthmus. X-rays of the thoracic spine showed a compression fracture of Th5 and Th7. There was no evident preoperative paraplegia. At surgery the aorta was completely transected. The aorta was crossclamped for 30 minutes without the use of a shunt and a graft was inserted. Three intercostal arteries were ligated. A short period of systolic hypotension (50 mmHg) occurred peroperatively. Postoperatively, paraplegia at the level of Th8 was evident.

Postoperative complications were the reason for death in 13 patients. These complications will be discussed in chapter VI.
The long term suvival rate after an operation for TAR was 30/46 (65%). Overall, the survival rate for all TAR patients was 30/57 (53%). The hospital stay after operation varied between 6 and 108 days, with a mean of 43 days. Many patients were referred for further revalidation to extended care facilities.

V.4. **Discussion**

In agreement with most reported series (a.o. Symbas, 1972; Kirsh et al., 1976; Ayella et al., 1977) the most frequent site of aortic rupture was the aortic isthmus. Surgical repair of lesions at that level posed no technical problems. Lesions extending to or mainly situated at the level of the aortic arch were more difficult to operate upon: two of three patients with TAR in this position were lost peroperatively. It seems important to localise the lesion exactly with preoperative angiography in order to be able to change the operative approach or the type of bypass if an aneurysm is present in a non classical location. Information about the status of brachio-cephalic vessels is of importance, as lesions of these vessels coexisted in 3 patients.
In 26/51 patients a complete transection of intima and media, often with separation of the two ends existed. Although some authors (Bromley et al., 1965; McClenathan and Brettschneider, 1965; Alley et al., 1966; Beall et al., 1969; Fontan et al., 1972; McCough and Hughes, 1973) favour the use of a direct anastomosis in repairing aortic tears, direct repair was possible in only four of our patients. In all other patients, a graft (n = 36) or patch (n = 2) was used. A variety of bypass techniques was used throughout the study; in the Radboud Hospital there has been a shift from the use of the LHB technique to simple crossclamping, in the University Hospital of Groningen to the TDMAC shunt. Due to heparinisation, two patients were lost because of bleeding; the role of heparinisation in the aggravation of CNS trauma could not be evaluated with certainty; in the crossclamping group, three patients developed postoperative paraplegia. One patient developed paraplegia in the LHB

group. Although the operations were uncomplicated, the ligation of the three intercostal arteries could have induced the paraplegia in two patients. In one of the patients a short period of peroperative hypotension was noted. The ethiology of the paraplegia in the other patient is not clear. In the patient from the LHB group who developed paraplegia, peroperative hypotension probably was the cause of the postoperative paraplegia. In the literature a shift exists from LHB and non heparinised shunts to 'simple' crossclapming of the aorta; the procedure seems however not without risk as can be seen in our - although small - group of patients. Other complications directly related to the bypass techniques or crossclamping did not occur. It is not yet clear from our data nor from the literature (Appelbaum et al., 1975; Kirsh et al., 1976; Akins et al., 1981) whether simple crossclamping - with reduction in operation time and no risk for bleeding disturbances - or a heparinised shunt without systemic heparinisation are to be preferred. However the three cases of paraplegia in the crossclamping group (n = 11) do not favour this technique in repairing TAR.

The type of shunt used, can influence the sequence of operations; only in frank bleeding should other operations (abdominal, cerebral) be performed first with the use of TDMAC shunt or crossclamping; with systemic heparinisation bleeding from other organs is more critical. A laparatomy, an abdominal tap or an ultrasonographic or CT-examination should resolve this problem (Aycila et al. 1977).

If other operations have priority, systolic tension should be kept below 110-120 mmHg, in order to prevent excessive strain upon the damaged segment of te aorta (Kirsh et al., 1976; Akins et al., 1981). An alternative approach was offered by Akins et al. (1981). In 14 patients elective delay (2-79 days) in the operation for TAR was chosen because of severe CNS trauma, extensive body surface burns, large contaminated open wounds, sepsis and severe respiratory insufficiency. Twelve of the 14 patients survived with antihypertensive treatment in which the systolic tension was settled between 100 and 120 mmHg. No experience with this approach, which can be useful in selected cases, exists in the Radboud Hospital and the University Hospital of Groningen.

Of the postoperative complications and deaths, none were directly related to the repair of the rupture except for the patient who was operated upon three times for hemomediastinum.

The other complications were due to the injuries of thoracic or abdominal organs, 'usual' postoperative complications like pulmonary embolism, or to a sequel of the status of the patient before operation (anuria as a consequence of shock). A surprising low number of cases of ARDS is found in our series, despite the often present pulmonary contusion (n = 22) and multiple extremity fractures (n = 38). Probably one of the reasons for this low frequency of ARDS is early fixation of extremity fractures and 'prophylactic' ventilatory assistance (Goris, 1983). Post surgical long-term survival after TAR operation was 30/46 (65%). Survival will be discussed under the heading VI.3.1.

PROGNOSIS OF PATIENTS WITH TRAUMATIC AORTIC RUPTURE

VI.I. **Introduction**

Longterm survival after traumatic aortic rupture without treatment is rare. From the data of Strassmann (1947) and Parmley et al. (1958) the natural history of TAR before operative treatment was possible, is known: of the patients arriving alive at the hospital, 25-30% die within 24 hours, 66% will be dead after 2 weeks, 82% after three weeks, and 90-95% within three months. Longterm survival with formation of a chronic aneurysm occurs in 2-5% of the reported cases.
Since operative treatment for TAR has become possible (Passaro and Pace, 1959), prognosis for TAR is improved. We will analyse the factors important for survival and survival rates.

VI.2. **Literature**

VI.2.1. *Survival*

In the light of the severe nature of most injuries causing TAR and the concomitant injuries, it is surprising to find, that with adequate treatment the percentage of overall survival is rather high. Table VI.1 shows data from the literature.

Table VI.1 Overall survival of patients with TAR who were operated upon

Year	*Author*	*Nr. patients*	
1971	DeMeules et al.	12	34%
1974	Fleming and Green	10	80%
1975	Appelbaum et al.	18	75%
1977	Ayella et al.	36	75%
1977	Bodily et al.	39	46%
1977	Pickard et al.	22	45%
1977	Turney et al.	31	81%
1977	Vasko et al.	43	60%
1978	Lacquet et al.	9	78%
1979	Richardson et al.	29	70%
1981	Akins et al.	44	75%
1983	Pinet et al.	52	64%

VI.2.2. *Analysis of fatal outcome*

The prognosis of the patients ariving alive in the hospital with TAR depends mainly on the following four factors:

1. time factor
2. influence of other lesions
3. sequence of operations
4. type of shunt used

VI.2.2.1. *Time factor*

It is evident that (missed) diagnosis and the time elapsed before an operation takes place, influences the prognosis.

Of 128 patients collected by Binet et al. (1962), 71 lived for 10 or more days. Heinrichs and Schwerd (1963) reported 47 patients studied at autopsy. Survival was one hour in 23 cases, 24 hours in 16 cases and 10 days in 8 cases. Jahnke et al. (1964) collected data from the literature concerning 38 patients who reached the hospital alive. They found that only 7 patients died within 12 hours after admission; the remaining patients died between 12 hours and 4 days after admission.

DeMeules et al. (1971) reported 7 deaths within 1-36 hours in all 7 patients in whom an unrecognised aortic rupture was present. Of Keen's (1972) 13 patients with TAR, 4 died within one hour of admission and 2 died, undiagnosed, 9 and 15 days after admission. Wilson et al. (1972) stated: 'the mortality rate for the first 24 hours after reaching the hospital is 25-50%, however if the patient lives for 24 hours after the accident, each day thereafter, he probably has a 90-95% chance for surviving at least another day without repair of the aorta. It can be justified to observe patients who are at very poor risk, because of severe associated injuries for the first 24 hours'. Prognosis without therapy is not much better now. Of 37 cases of TAR (Sevitt, 1977) 25 lived up to 13 days (20-13 days). Hemorrhage from rupture was judged causal or contributary to death in 20 patients.Bodily et al. (1977) missed the diagnosis in 5 of 26 potentially salvageable TAR patients; exsanguination occurred between 12-36 hours after admission. Several authors (Keen 1972, O'Sullivan et al., 1972, Kirsh et al., 1976, Pickard et al., 1977, Plume and DeWeese, 1979) reported a mortality of 8-40% before operation, either in the emergency room or at the radiology department due to exsanguination from the aortic rupture. Only the report of Vasko et al. (1977) contradicts this experience. They stated: 'delay in recognition and operation did not appear to influence morbidity and mortality unless hemodynamic instability or renal impairment was present'.

Akins et al. (1981) applied pharmacological therapy with various agents to lower the blood pressure (propanolol, alpha-methyl-dopa, trimethaphan, nitroprusside, hydralazine and hydrochlorotiazade) to perform elective operation in 14 patients with TAR. The delay for the operation was 2-79 days. Reasons for the delay of the

operation were evaluation and stabilisation of extensive CNS traumata, extensive body surface burns that have a high risk of infection, large, contaminated open wounds, established sepsis and severe respiratory insufficiency. There was a 86% survival in this group. Five other patients had surgery deferred indefinitely because of severe CNS trauma; there were no deaths during pharmacotherapy with a mean follow-up of 51 months. The importance of lowering the blood pressure when an operation has to be delayed seems obvious from this data.

VI.2.2.2. *Influence of other traumatic lesions*

There is quite a difference in the various reports concerning the incidence of concomitant (lethal) injuries in patients with TAR. Greendyke (1966) reported potential lethal injuries to be present in 28 of 42 (67%) patients with TAR, however this was an autopsy study. DeMeules et al. (1971) operated upon 8 patients with TAR, 4 died of concomitant abdominal or intra-cerebral injury. Concomitant lethal injuries were present in 3 of 31 patients reported by Turney et al. (1976). Of the 12 patients with abdominal injury in Kirsh's series (1976), 6 first underwent a celiotomy and five of them died. Bodily et al. (1977) reported concomitant lethal injuries in one third of his 39 patients with TAR. These 13 patients suffered 62 major concomitant injuries (average 4.8/patient) Ten patients died during transport to the hospital or at the time of cardiopulmonary resuscitation after admission; 3 died because of intraabdominal hemorrhage in spite of an emergency laparotomy. Exsanguination of 8 of the 13 patients was not due to aortic rupture but to other causes (CNS injury, intraabdominal bleeding).

VI.2.2.3. *Sequence of operations*

The decision whether to perform a celiotomy or thoracotomy first must fit the circumstances. Kirsh et al. (1970, 1976) stated 'the fact that an untreated aortic rupture follows such an unpredictable course, makes us feel that aside from rapidly progressing cerebral injuries or massive intra-abdominal hemorrhage, traumatic rupture of the aorta deserves the highest priority and its repair should be carried out first'. Repair of intra-abdominal injuries was undertaken in 6 patients. Five died due to exsanguination before thoracotomy could be performed.

VI.2.2.4. *Influence of the type of shunt*

Table VI.2 compares mortality rates of bypass versus no bypass technique in recently reported series in the literature (Appelbaum et al., 1975; Kirsh et al., 1976; Turney et al., 1977; Vasko et al., 1977; Akins et al., 1981).
Although probably not totally comparable, these data continue to support the trend away from the left heart bypass. Akins et al. (1981) however prefer to use a

Table VI.2 Mortality rate and methods to prevent distal ischemia in the recent literature

	Nr. patients	*Nr. deaths*
Left heart bypass	56	23
External shunt	66	8
No shunt	19	0

shunt, because in a training hospital crossclamping cannot always be limited to less than 30-45 minutes.

VI.3. **Own patients (Nijmegen and Groningen)**

VI.3.1. *Survival*

The fate of patients with TAR is influenced by many factors. If the patient survives the initial trauma, early detection and subsequent operation are of vital importance for survival. The role of concomitant trauma is discussed in chapter IV. In this chapter we give an analysis of the fatal outcome in our patient population (see table VI.3).

Table VI.3 Outcome of our patients with acute TAR (n = 57)

	Nr. patients	
Not detected on admission; death	1	2%
Death before angiography	3	5%
Death after angiography and before the first operation	4	7%
Death during celiotomy, before TAR repair	2	4%
Death during operation for TAR	4	8%
Not operated for TAR	1	2%
Death after the operation for TAR	13	23%
Long-term survival after surgery for TAR	30/46	65%
Overall survival (acute group)	30/57	53%

VI.3.2. *Analysis of fatal outcome*

VI.3.2.1. *Time factor*

Our total group of patients with TAR consists of 68 patients. In 11 patients, TAR was not detected initially at admission and these patients developed a chronic

aneurysm (chapter VIII). Ten patients died before thoracotomy, 4 patients died during the operation and 13 after the operation for aortic repair (Table VI.3).
In only two patients, TAR was not diagnosed before death: one patient died one hour after admission; autopsy revealed a ruptured aorta at the isthmus. The findings on the chest X ray of this patient were rather subtle and had not raised the suspicion of TAR (Fig. III.14); the other patient died due to an unrecognised aortic rupture during celiotomy (Fig. III.15). Three patients - in which TAR was suspected from the chest roentgenogram - died respectively 1 hour, 4 and 12 hours after after admission before angiography could be carried out. Four patients died after angiography, before operation. Of these, three patients died from rupture respectively 2, 6 and 24 hours after admission; one patient was decleared dead 36 hours after admission due to cerebral trauma. Table VI.4 summarizes the findings.

Table VI.4 Death in TAR patients before operation (n = 10) for TAR

Cause of death	*Nr patients*	*Interval trauma-death in hours*
Rupture (not recognised)	1	1
Rupture before angiography	3	1, 4, 12
Rupture after angiography before one operation	3	2, 6, 24
Cerebral death after angiography	1	36
At celiotomy, before TAR repair	2	3, 8

VI.3.2.2. *Influence of other traumatic lesions*

Two patients died from postoperative complications not related to concomitant injuries: one on the 11th postoperative day due to massive pulmonary embolism; another patient sustained damage of the innominate artery as a complication of the tracheostomy and prolonged ventilation with a tracheostomy tube. Erosion of the innominate artery was treated primarily by reconstruction, but repeated bleeding necessitated ligation of the artery. In this patient infection (mediastinitis and widespread sepsis) was the cause of death 6 weeks postoperatively. Two patients died postoperatively form bleeding due to systemic heparinisation. Nine patients died

Table VI.5 Postoperative death due to concomitant trauma (n = 9) in patients with TAR

Cause of death	*Nr. patients*	*Days between operation and death*
CNS trauma	5	2, 3, 4, 7, 8
Sepsis	4	6, 28, 41, 70

postoperatively due to concomitant injuries (table VI.5). In 25 patients it was possible to obtain an injury severity scale (I.S.S.), Goris (1983). In 12 patients of the survival group the I.S.S. was on the average 44; in 12 patients with a fatal outcome the I.S.S. was on the average 53, ($p < .002$).

VI.3.2.3 *Sequence of operations*

Of the 49 patients operated upon, 28 first underwent repair of the aorta, in 17 patients a celiotomy was performed first. A craniotomy was performed as first operation in three patients: all three patients died after repair of the TAR. Osteosynthesis of extremity fractures was performed as the first operation in one patient; suspicion of TAR was raised three days later. Survival of 28 patients who were operated upon first for their aneurysm was 19/28 (68%); survival of 17 patients who underwent a celiotomy first was 10/17 (58%), ($p > 0.71$).
Of the 17 patients in whom a celiotomy was first performed 7 died: one peroperative from liver and spleen injuries, and one patient due to aortic rupture. The other five patients died from various causes. Of our eleven patients where a celiotomy was performed after aortic repair, two patients died: one due to massive intra-abdominal bleeding from heparinisation, the other due to sepsis.

VI.3.2.4 *Influence of type of shunt*

In two patients death was related to the systemic heparinisation used with LHB shunt. One death was caused by excessive bleeding from pelvic veins. During the aortic repair, massive blood loss into the abdomen was noted; during celiotomy, 10-15 litres of blood were found in the abdomen. Bleeding from pelvic veins could not be stopped and the patient died. The other patient developed per- and postoperatively a coagulopathia, probably related to the systemic heparinisation. This coagulopathia could not be reversed and the patient died from diffuse intraabdominal bleeding. There were no per- or postoperative deaths related to TDMAC shunt or crossclamping.

VI.3.3. *Autopsy findings*

Autopsy was performed in nine patients. In the autopsy of the five patients who died before an operation was performed (one missed diagnosis, four deaths before angiography could be performed) rupture of the false aneurysm in the pleural space was the cause of death.
In four patients who died postoperatively, autopsy was performed. In all of these cases the sutures were intact. In one patient (born 1897) cardiomyopathy and pleuritis were reported; in one patient pleuritis and mediastinitis were present due

to multiple operations on the innominate artery; one patient died from cerebral infarcts; in one patient a massive pulmonary embolus was detected as the cause of death.No major unsuspected injuries of abdominal or other organs were detected at autopsy.

VI.4. **Discussion**

As can be seen from table VI.3, early mortality remains a serious problem in patients with TAR. 10/57 (17%) of our patients died before an operation for TAR could be performed, all except one due to rupture of the false aneurysm into the pleural space. Our experience is similar to that of others (Keen, 1972; O'Sullivan et al., 1972; Wilson et al., 1972; Bodily et al., 1977; Pickard et al., 1977; Plume and deWeese 1979), who report an early mortality in the first 24 hours of 8-50%. Only Vasko et al. (1977) reported that delay in recognition of TAR and operation did not appear to influence morbidity and mortality.

On the other hand, in our series, 14 patients were operated 3-6 hours after trauma, 17 between 6 and 24 hours, and 9 later than 24 hours. Although these patients were relatively stable, the problem is that one can never predict in which patient fatal rupture will occur. It is obvious that angiography and surgery must be performed as soon as possible in patients with TAR.

Contrary to some other countries, it is not obligatory in The Netherlands to perform an autopsy after lethal traumatic injuries. For this reason we do not know for sure if our two patients with a missed diagnosis of TAR are representative or an underestimation of the number of missed diagnosis. It is clear that failure to recognise TAR, seriously endangers the patient. We had no experience with elective operations under anti-hypertensive treatment as reported by Akins et al. (1981).

In 9/57 (16%) concomitant injuries were the direct or indirect cause of postoperative death (table VI.5). In 5 patients, CNS trauma was the main cause of death. Widespread sepsis leading to death in 4 patients originated from concomitant injuries to abdominal organs or lungs. Bleeding from lacerated abdominal organs (liver, pelvic veins, spleen) were the reason for peroperative and postoperative mortality in two patients. As expected, other organ lesions have a profound influence on prognosis, especially intracerebral and abdominal injuries.

DeMeules et al. (1971) and Bodily et al. (1977) reported a higher death percentage (50 and 34% respectively) in TAR patients when concomitant injuries were present. Our data are comparable with those of Kirsh et al.(1976) and Turney et al. (1977), who reported a concomitant injury/mortality rate of 8 - 10%.

As regard to the sequence of operations, no complete ruptures of the traumatised aorta occurred in our patients in whom a celiotomy was performed first, with the exception of one patient who died from an unrecognised rupture while diaphragmatic repair was undertaken. Kirsh et al. (1976) lost 5 of 6 patients undergoing a

celiotomy before aortic repair due to exsanguination. The reason for performing a celiotomy first was frank abdominal bleeding or clinical or angiographically suspected laceration of spleen or liver in stable patients, in whom left heart bypass with systemic heparinisation was planned. The effect of systemic heparinisation was a direct cause of death in 2 patients. In three patients with CNS trauma, heparinisation could have influenced prognosis. Heparinisation was the cause of intracerebral bleeding and subsequent death in two patients of Kirsh et al. (1976). All of the deaths in Vasko's et al. (1977) patients were directly or indirectly the result of excessive bleeding and occurred in patients who had been heparinised. The risk of bleeding due to heparinisation in multiple injured patients is stressed by several authors (Kirsh et al., 1976, Vasko et al., 1977).

When abdominal or cerebral injury without evident bleeding is present, it is justifiable to repair first the aortic tear with a shunt not requiring heparinisation or by crossclamping and to perform a celiotomy as second operation.

Autopsy revealed rupture of the aortic tear in the pleural space as the cause of death in 5 patients. In 2 patients an unsuspected aortic rupture was detected at autopsy. In one case a massive pulmonary embolus was detected as the cause of death. No other major - clinical unsuspected-injuries were detected at autopsy. Unfortunately, in The Netherlands autopsy is performed rather infrequently in trauma patients with lethal injury; more exact information regarding the spectrum of injuries and the cause of death could be very stimulating for the physician involved in trauma care.

Our long term survival rate was (30/46) 65% for the patients operated upon for TAR; the overall survival was (30/57) 53%. The deaths can be categorised as follows: missed diagnosis 1/57 (2%), rupture before operation of TAR 7/57 (12%), peroperative death 6/57 (11%) and postoperative death 13/57 (23%), (Table VI.3). Our survival rate of the patients operated upon (65%) is somewhat higher as compared to the reports of DeMeules et al. (1977), Pickard et al. (1977), and lower than in the review of Fleming and Green (1974), Ayella et al. (1977), Turney et al. (1977), Akins et al. (1981). Our survival rate is comparable with the results of Pinet et al. (1983), (table VI.1).

It is clear from the literature and from our experience that prognosis with TAR is influenced mainly by:

1. correct diagnosis
2. time elapsed between admittance and operation
3. localisation of the rupture
4. concomitant injuries
5. protection method.

Part B

CHRONIC TRAUMATIC ANEURYSMS OF THE THORACIC AORTA

Chapter VII

GENERAL ASPECTS OF CHRONIC TRAUMATIC ANEURYSMS OF THE THORACIC AORTA

VII.1. **Introduction**

Chronic posttraumatic aneurysms (CTAR) of the aorta are uncommon. An aneurysm is defined as chronic when older than 3 months (Finkelmeyer et al., 1982). Approximately 2-5% of patients with TAR survive without treatment and develop a chronic posttraumatic aneurysm (Parmley et al., 1958; McBurney and Vaughan, 1961; Bennett and Cherry, 1967). Chronic posttraumatic aneurysms comprise 2-28% of all thoracic aneurysms (DeBakey et al., 1958; Dubost et al., 1962; Joyce et al., 1964; Schonholtz and Hahnke, 1964; McClenathan and Brettschneider, 1965). For survival and with the formation of a CTAR, either the adventitial wall must be preserved or the surrounding tissues must be able to contain the pulsating hematoma that forms after the layers of the aortic wall are torn.

Complete transection of the aorta with separation of the two ends does not preclude longterm survival; 5 of 6 cases reported by Groves (1964) showed this type of injury, and Parmley et al. (1958) have made a similar observation. While the most acute danger for rupture exists within the first three weeks, the degree of fibroplasia in the wall of the false aneurysm is still insufficient to provide much garantee against rupture for at least three months (Groves, 1964). Fatal rupture can still occur 1 to 3 months after injury (Parmley et al., 1958).

Regarding the ethiology of chronic posttraumatic aneurysms: most patients were involved in automobile accidents (Spencer et al., 1961; Finkelmeyer et al., 1982). The diagnosis of a traumatic aneurysm at the time of the initial trauma was missed in all reported patients although most patients were hospitalised and chest X-rays were taken. One of the interesting findings in the series of Fleming and Green (1974) was that most patients with a CTAR had no significant abdominal injuries at the time of the initial trauma. Most patients were discharged in good health and presented themselves after a variable time with symptoms due to the CTAR or were detected by chance.

VII.2. **History**

The first operations for chronic traumatic aneurysms of the aorta were performed in the early 1950's (Weisel et al., 1951; Banhson, 1952; Stranahan et al., 1953); however until the introduction of partial cardiac bypass techniques (Cooley et al., 1957;

Gerbode et al., 1957) an operation for CTAR was associated with such high morbidity and mortality rates, that a philosophy of conservative management prevailed among thoracic surgeons, unless enlargement or symptoms were present(Hollingsworth et al., 1952; Goyette et al., 1954; Eiseman and Rainer, 1958).
Fleming and Green (1974) stated: 'a review of the American literature prior to 1957 revealed that only 21 patients with CTAR of the thoracic aorta have been reported: two of them died from rupture prior to an operation; one patient's aneurysm ruptured and he died during an emergency operation; one patient died intraoperatively; 11 were successfully treated and 6 were followed conservatively'. However, with advances in vascular surgery , elective resection of a thoracic aneurysm was no longer associated with a prohibitive mortality. DeBakey et al. (1958) reported that their group had resected 60 chronic traumatic aneurysms without mortality. McClenathan and Brettschneider (1965) reported eleven successful operated cases. In The Netherlands, the first publication for an operated CTAR is from the de Boer and Kwan (1971).

VII.3. **Frequency of occurrence**

These data were given in the introduction. Probably today more cases are seen than reported in the fifties and sixties as there has been a tremendous rise in traffic accidents with chest trauma. Finkelmeyer et al.(1982) reported 413 cases in a collective review of the literature between 1950 - 1980.

VII.4. **Pathology**

Since most traumatic aortic ruptures occur at the aortic isthmus, it is reasonable to expect that most chronic traumatic aneurysms will arise at the same site. 94% of CTAR were localised at the isthmus in a review of 413 patients Finkelmeyer et al. (1982). If the patient survives for several weeks a fibrous wall is formed, which provides a greater degree of stability. The time required to convert the pulsating hematoma into an aneurysm must be extremely variable, but Groves (1964) observed such a transformation as early as 23 days after trauma. He found that the false aneurysm wall connecting the aortic ends was composed of tissue of various thickness and frequently could not be separated from the adjacent viscera (the tracheobronchial tree and the esophagus), which were actually part of the wall. According to Bennett and Cherry (1957), the aneurysm is often calcified and thrombosis may occur in the false aneurysm and may even partially obstruct the aortic lumen. Dotter (1961) stated: 'the rate of aneurysmal dilatation varies with a number of factors, the most important being the size and extent of the initial hemorrhage into the aortic wall, the extent of destruction of smooth medial muscle and elastic tissue,

the thickness, strength and distribution of reactive fibrous tissue at the outer surface of the aortic wall and the intraluminal pressure'. Garamella et al. (1962) reported marked thickening and fibrosis of media and adventitia in their cases of CTAR. Sometimes erosion of vertebral bodies or even fistulae to the esophagus were found. Groves (1964) stated: 'in every case I was impressed by the thinness of the aneurysmal wall, being composed of fibrous tissue without elastic tissue'. According to Molnar and Pace (1966) most chronic aneurysms were saccular with only partial rupture of the aortic wall. A fusiform aneurysm is present if complete transection of the aortic wall takes place. Complete transection of the aorta was found in 14/33 (42%) of the chronic aneurysms (Fleming and Green, 1974).

VII.5. **Natural history**

Connolly (1962), Groves (1964), Joyce et al. (1964) and McClenathan and Brettschneider (1965) believe that traumatic thoracic aortic aneurysms have a similar natural history as syphilitic or arteriosclerotic aneurysms. In a five year follow-up study of 97 unclassified thoracic aortic aneurysms before the time of excisional surgery, DeBakey et al. (1958) reported that 31 patients had died, the majority from rupture of the aneurysm. The probability of enlargement and rupture makes elective surgery mandatory in good risk patients. In the past this point has been disputed, especially by Steinberg (1957), who stated that the risk for rupture of an asymptomatic traumatic aneurysm of long duration is small and that surgical intervention is not always necessary. However, McClenathan and Brettschneider (1965) have demonstrated that Reynold's number of laminar flow is exceeded in the region of the aneurysm and turbulence occurs. Resistance to flow and the energy required to maintain a given flow also increases. As velocity through the aneurysm decreases, lateral pressure on the wall increases according to Bernoullies principle. Further, as the diameter of the aneurysm increases, the wall tension is greater with the same intraluminal pressure according to La Place's equation. These mechanical and hemodynamic factors dictate surgical rather than expectant therapy (Schwartz et al., 1975).

Clinical experience is summarized by Bennett and Cherry (1967). They reviewed the literature from 1950-1965 and collected 105 chronic traumatic aneurysms. A study of these cases provided little support for the concept that chronic traumatic aneurysms are stable.Only 41% of the aneurysms were stable with absence of enlargement or symptoms. Radiological proven enlargement was present in 21% of the cases; fifty percent of the patients developed symptoms attributable to the aneurysm. A symptom-free period of months or years following the injury is common for patients with chronic traumatic aneurysms. Of 105 cases, 22% were aymptomatic for a period of more than five years, but manifested symptoms or radiological evidence of expansion later. Bennett and Cherry (1967) stated: 'from a study of

the literature it can be concluded that there is no way to predict which aneurysms will undergo enlargement and which will remain quiescent.'

In the review of Finkelmeyer et al.(1982) of 413 cases collected from the literature between 1950-1980, 65% were symptomatic at the time of the operation. Nineteen percent of their patients showed enlargement of the aneurysm on serial roentgenology. The longest interval between trauma and rupture is reported by Eiseman and Rainer (1958) in a patient whose aneurysm was stable for 43 years and subsequently enlarged during the following 4 years. Another case was described by Ricen and Dickens (1942); a patient, asymptomatic for 27 years died suddenly of rupture of the aneurysm after 8 days of pain and fever. Seventeen percent of the patients with a chronic traumatic aneurysm reported since 1950 first developed symptoms or enlargement 10 years after the rupture (Bennett and Cherry, 1967).There are however several case reports of aneurysms rupturing after 3 months (Jay, 1954; Binet et al., 1961; McBurney and Vaughan, 1961; Garamella et al., 1962; Groves, 1964).

Some cases of bacterial infection of chronic traumatic aneurysms are known (Strijker, 1948; Garamella et al., 1962;Gwathmey and Byrd, 1964).

In 1974, Fleming and Green reported 33 patients with a chronic traumatic aneurysm from the Walter Reed Army Medical Center. Automobile accidents were the cause of the lesion in over 90% of the cases. An interesting finding in their study was that there were no severe abdominal injuries at the time of the initial trauma in their patients with chronic traumatic aneurysm. An increase in size was detected in 18%; 30% of the patients developed symptoms 10 years after injury. Overall, 39% of the patients showed signs of instability manifested by symptoms, increasing size of the aneurysm or both. During the first three years, 7 out of 14 patients had symptoms due to the aneurysm. Of 15 patients who underwent operative repair more than five years after trauma, 6 (40%) had symptoms or radiological proven enlargement of the aneurysm. The mean interval between trauma and operation was 6.7 years (110 days-21 years). Of 60 patients with CTAR not subjected to operative treatment, compiled by Finkelmeyer et al, (1982),20 (30%), died during the period in which they were followed due to their aortic lesions. Most deaths were due to sudden rupture of the aneurysm. Twenty years after the injury, there was only a 33% probability of being alive and free of signs or symptoms of aneurysm expansion. Patients with CTAR not subjected to operative treatment demonstrated a lower probability of survival than did the operated group.

VII.6. **Diagnosis**

Patients with chronic aortic rupture are detected because of clinical symptoms or because they have a chest film for unrelated reasons.

VII.6.1. *Clinical symptoms*

Because most posttraumatic chronic aneurysms are located at the isthmus in proximity of the left main stem bronchus, the recurrent laryngeal nerve and the esophagus, symptoms such as dyspnea, cough, hoarseness and dysphagia are easily explained. Symptoms were present in 53% of the patients of Bennett and Cherry's series (1967) and in 65% in Finkelmeyer's et al. series (1982). Pain was the leading symptom in one third of their patients, presumably due to compression or stretching of the viscera and nerves in the vicinity of the aneurysm. The onset of symptoms is indicative of the expansion of the aneurysmal sac (Steinberg, 1957). Murmurs are described in 40% of chronic posttraumatic aneurysms (Fleming and Green, 1974). The fact that symptoms develop and increase in severity with time and that additional ones appear is striking and is further evidence for the progressive enlargement of the aneurysm which so frequently occurs (Groves, 1964; Bennett and Cherry, 1967; Fleming and Green, 1974; Finkelmeyer et al., 1982). Spencer et al. (1961) reported eight patients with chronic traumatic aneurysm, three were asymptomatic, five had symptoms, three of whom had chest pain, one a slight cough and one severe dyspnea. Appelbaum et al. (1975) observed seven patients 2-15 years after trauma with a chronic traumatic aneurysm. Three patients had increasing back pain, one had progressive dysphagia and one hoarseness from recurrent laryngeal nerve involvement. At operation the aorta was completely transected in all patients. Of eight patients reported by Vasko et al. (1977), 4 complained of chest pain and one of dysphagia. Of the 20 patients reported by Soyer et al. (1981), 9 were symptomatic. In one of their patients the aneurysm was a source of emboli.

VII.6.2. *Chest X-ray findings*

A widened mediastinal shadow due to the initial mediastinal hematoma can develop into a localised bulge in the descending aorta just below the origin of the left subclavian artery (Hollingsworth et al., 1952; Wyman, 1953; Goyette et al., 1954; Fishbone et al., 1973).

In the chronic phase, a well circumscribed mass in the region of the aortic knob can be seen. This mass may be pulsatile. On the chest X-ray of patients with a chronic traumatic aneurysm of long duration often calcium deposits in the wall can be seen (Steinberg, 1957; Fleming and Green, 1974). Progressive enlargement may be seen on serial films (Fleming and Green, 1974). A chronic traumatic aneurysm must be differentiated from other aneurysms (syphilitic, arteriosclerotic, congenital, mycotic) in the proximal descending aorta. The syphilitic type is usually associated with aortitis; an irregular, dilated and calcified ascending aorta often accompanies a syphilitic aneurysm. A positive serologic test for syphilis is confirmatory (Steinberg, 1957). In atherosclerotic aneurysms, there are frequently other signs of atherosclе-

rosis. A history of a deceleration trauma and the age of the patient are other differentiating factors. Tumors of the mediastinum are readily differentiated by other radiologic methods (tomography, angiography, CT). Not infrequently, chronic traumatic aneurysms are detected by chest X-rays taken for unrelated reasons.

VII.6.3. *Angiography*

The chronic posttraumatic aneurysm of the aorta is usually of the saccular type arising from the anterior wall of the aortic isthmus. The posterior wall of the isthmus usually has a normal contour. The lumen of the aneurysm may be much smaller than what is visualised on the plain film due to the presence of an organised trombus (Fishbone et al., 1973).

VII.7. **Surgery**

The first operations for chronic traumatic aneurysms of the aorta were performed in the early fifties (see introduction). With the advent of the cardio-pulmonary bypass techniques (Cooley et al., 1957; Gerbode et al., 1957) and advances in vascular surgery and anesthesia, elective resection of a chronic posttraumatic aneurysm of the aorta was no longer associated with a prohibitive mortality. Cooley (1964) stated that his group had resected chronic traumatic aneurysms in 60 patients without mortality.

Fleming and Green (1974) reported a mortality rate of 3.5% in 33 patients operated upon for chronic traumatic aneurysms. The fact that these chronic aneurysms are usually found in young and otherwise healthy persons, free of degenerative vascular disease, explains the low surgical mortality rate. Because of these low mortality rates, there has been increasing endorsement of elective surgery for all cases of chronic traumatic aneurysm even the calcified, asymptomatic lesions of many years duration (Dubost et al., 1961; Spencer et al., 1961; Connolly, 1962; Groves, 1964; McClenathan and Brettschneider, 1965).

Surgical techniques and prevention of distal ischemia are the same as in case of acute aortic rupture (these are discussed in chapter V). Mortality rates related to operations for chronic traumatic aneurysms are low. Cooley (1964) reported 0% in 60 patients, Heberer (1971), 6% in 16 patients and Fleming and Green (1974) 3.5% in 33 patients. Finkelmeyer et al. (1982) reported in an review article a 4.6% mortality in 332 patients operated upon for CTAR.

OWN STUDY: CLINICAL AND RADIOLOGICAL FINDINGS IN ELEVEN PATIENTS WITH CHRONIC TRAUMATIC ANEURYSM OF THE THORACIC AORTA

VIII.1. **Introduction**

In this chapter we will present the own experience with chronic traumatic aneurysms of the thoracic aorta. A retrospective analysis is performed on 9 patients with CTAR referred to the University Hospital in Nijmegen and 2 patients referred to the University Hospital of Groningen between 1971 and 1984. We present and analyse clinical and radiological data at the time of the first admission, where available, and at the time of detection of the aneurysm. We will compare and discuss our findings with those in the literature.

VIII.2. **Own patients**

Table VIII.1 provides data of the 11 patients with CTAR.
Four patients were female, seven male. The age at the time of the first admission varied between 16 and 39 years with an average of 25.2 years. At the time of detection, the age varied between 21-40 years with an average of 29.5 years. The

Table VIII.1 Data of the own patients with chronic traumatic aneurysm of the thoracic aorta

Patient Nr.	*Birth year*	*Sex*	*Year primary trauma*	*Year of detection*
1	1951	M	1967	1970
2	1941	M	1966	1971
3	1942	F	1970	1975
4	1946	F	1971	1977
5	1953	M	1972	1978
6	1927	M	?	1981
7	1940	F	1976	1981
8	1953	M	1971	1981
9	1944	M	1969	1981
10	1961	F	1982 (june)	1982 (oct.)
11	1943	M	1982 (july)	1983 (febr.)

time between the first and the second admission was on the average 5.3 years, with a range from 3 months to 12 years. The year of the primary trauma is listed in table VIII.1.

VIII.3. **Diagnosis**

VIII.3.1. *Clinical symptoms*

Four patients had no complaints at all at the time of detection of the aneurysm. Three patients had minimal and aspecific complaints: one had mild pain in both arms during work, one had a vague sensation of pressure at the thorax while stooping, and one patient showed aspecific thoracic pain two months after the injury. Of the four patients with more severe complaints, two showed symptoms of dysphagia, one patient had severe complaints of thoracic pain, back pain and hoarseness, and the last patient showed symptoms of dyspnea and recurrent infections of the left lung.
In only two patients a systolic murmur could be heard at the back. Fleming and Green (1974) reported a 40% incidence of systolic murmur in chronic posttraumatic aneurysm.

Manner in which the chronic traumatic aneurysm was detected
In three patients the aneurysm was detected during mass screening, in two on a preoperative chest X-ray, in one on a chest X-ray taken for unrelated reasons and in five patients, complaints were the reason for a thoracic roentgenogram. Table

Table VIII.2 Manner in which the chronic traumatic aneurysm of the aorta was detected

Patient	*Complaints*	*Mode of detection*
1	Arm pain during work	Mass screening
2	Thoracic pain while stooping	Mass screening
3	Dysphagia	Results of complaints
4	–	Pre-operative chest X-ray
5		Mass screening
6	Thoracic pain, hoarseness	Result of complaints
7		Pre-operative chest X-ray
8	–	Chest X-ray for unrelated reason
9	Dyspnea, recurrent infections	Result of complaints
10	Dysphagia	Result of complaints
11	Thoracic pain	Result of complaints

VIII.2 shows the above mentioned findings. Hypertension was present in two patients; the relation with the chronic traumatic aneurysm was not evident.

VIII.3.2. *Radiography*

VIII.3.2.1. *Radiography at the time of the initial trauma*

It was possible to review the admission chest radiographs of five patients (patient nr. 3, 7, 8, 10, 11). Table VIII.3 shows the signs that were present on these thoracic roentgenograms and fig VIII. 1 and 4 - 7 the radiographic findings of these patients.

Table VIII.3 Signs on the initial chest X-ray suggestive of aortic rupture in 5 patients with CTAR*

	Patient number				
Chest X-ray signs	*3*	*7*	*8*	*10*	*11*
Mediastinal width 6-8 cm	+		+		
Mediastinal width 8-10 cm		+		+	s
Tracheal displacement to the right	+	–	–	+	+
Downward displacement of the left main bronchus	–	–	–	+	+
Displacement of nasogastric tube	–	–	–	+	–
Unsharp aortic outline	+	+	+	+	+
Broadening of paratracheal stripe	+	+	+	+	+
Displacement of the right paraspinal line	+	–	–	?	–
Displacement of the left paraspinal line	+	–	–	+	–
Fracture of first or second rib	–	–	–	–	–
Other rib fractures	+	–	+	–	+
Hemothorax	+	–	+	–	+
Apical cap	+	+	?	+	–
Displacement of superior vena cava	–	–	+	–	–
Opacified pulmonary window	+	–	+	–	–

* Fig. VIII.1 and 4 - 7.

Retrospectively, there were many signs on the chest roentgenograms of these patients indicating a hemomediastinum and angiography should have been performed. Probably one of the major reasons that these patients had no further work up such as angiography is, that a more subtle evaluation of the thoracic roentgenogram of patients with blunt chest trauma was not usual in the late 1960's and early 1970's.

VIII 3 2 2 *Radiography at the time of detection of the chronic aneurysm*

The chest X-rays were made for various reasons at the time of detection (table VIII 2) In three patients abnormalities were detected at mass screening, in two patients the aneurysm was detected on a pre-operative evaluation, in one patient on chest roentgenograms taken for unrelated reasons Complaints were the reason for a thoracic roentgenographic examination in five patients Radiographic findings were rather subtle except for patients 1, 2, 5 and 6, where a large mass was seen in the region of the aortic knob (fig VIII 3 a) In the other patients a slight change in the contour at the proximal part of the descending aorta could be seen, twice with a thin clacified rim On the lateral chest film an abnormal density in the region of the proximal descending aorta or in the aorto-pulmonary window was seen in two patients (Pat 5, Pat 8), (Fig VIII 5 c) Old rib fractures were present in four patients In one patient enlargement of the aneurysm within a short time was evident Table VIII 4 summarizes the findings on the chest X-rays at the time of detection

Table VIII 4 Chest X-ray findings at the time of detection of the chronic traumatic aneurysm in 11 patients

Patient nr	*Chest X-ray findigns*
1	Mass density at the aortic knob
2	Mass density at the aortic knob + calcification
3	Slight contour abnormality below aortic knob + thin rim of calcification
4	Mass below aortic knob + thin rim of calcification/old rib fractures
5	Calcified mass at level of aortic knob
6	Large mass proximal descending aorta/old rib fractures
7	Abnormal aortic knob configuration
8	Abnormal aortic knob configuration/old rib fractures
9	Abnormal aortic knob configuration + rim of calcification
10	Slightly abnormal contour of the aortic knob/increased density in aorto-pulmonary window
11	Slightly abnormal contour of the aortic knob/old rib fractures

Examples of radiographic findings of patients with CTAR are given in the figures VIII 1 - 7

Other radiographic investigations

In two patients (patient 3 and 10) an X-ray examination of the esophagus was performed because of complaints of dysphagia One of these patients showed a slight, the other a rather large impression (fig VIII 6 c) on the esophagus caused by the false aneurysm In one patient, bronchography was performed which showed a

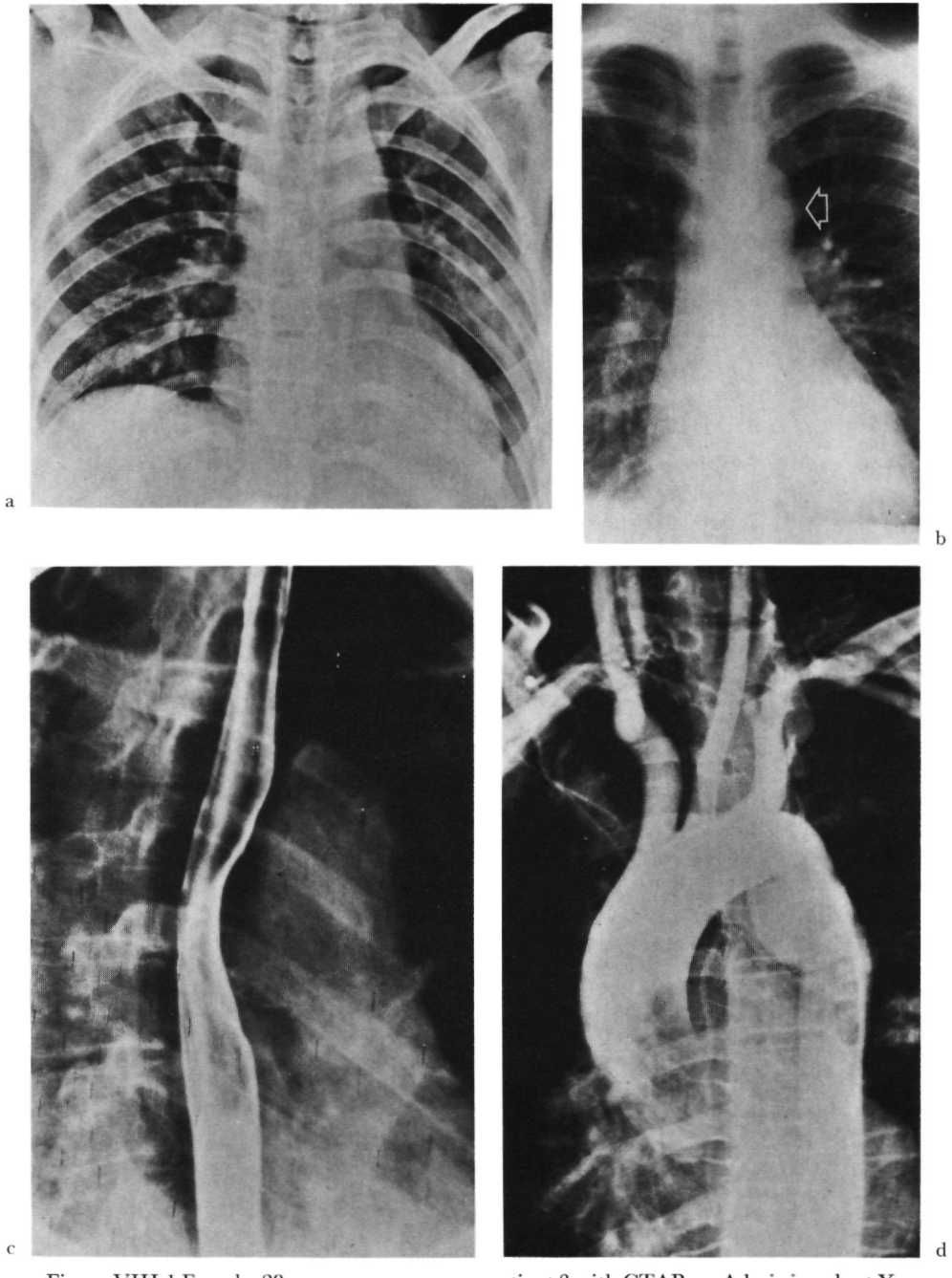
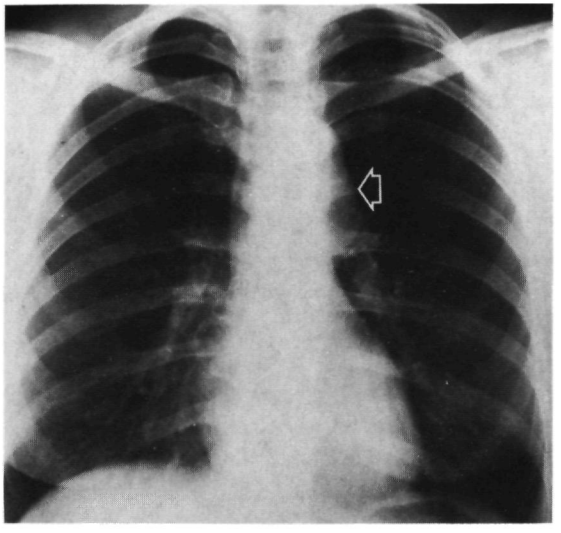


Figure VIII.1 Female, 28 years, car passenger, patient 3 with CTAR. a. Admission chest X-ray 03.05.70: unsharp aortic outline; discrete widening of the mediastinum (7 cm); normal tracheal position; left rib fractures 6 and 7. Diagnosis of TAR missed. b. Chest X-ray 16.10.75: abnormal contour of aortic knob with discrete calcification (white arrow). c. X-esophagus: impression at the level of the aortic knob. d. Angiography: false aneurysm at the aortic isthmus.



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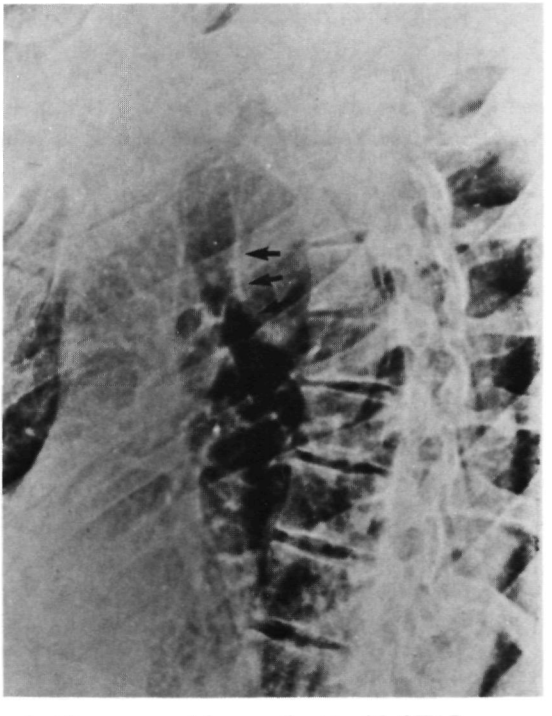
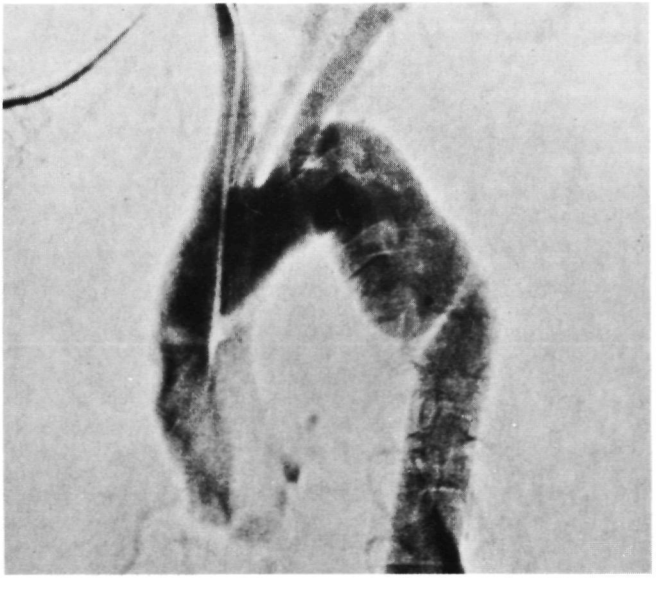


b

Figure VIII.2 Female, 23 years, car driver, patient 4 with CTAR.
a. and b. Frontal and lateral chest X-ray at the time of detection (1977): abnormal aortic contour at the level of the aortic knob (arrow). An impression on the tracheal wall is visible on the lateral chest X-ray (arrows). Primary trauma 1971. c. Femoral angiography: the aneurysm could not be passed, catheter in the false aneurysm. d. Complementary brachial arteriography: false aneurysm at the level of the aortic isthmus.

c



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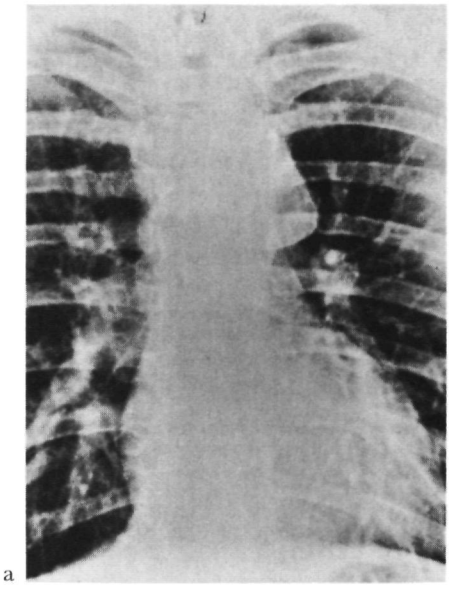

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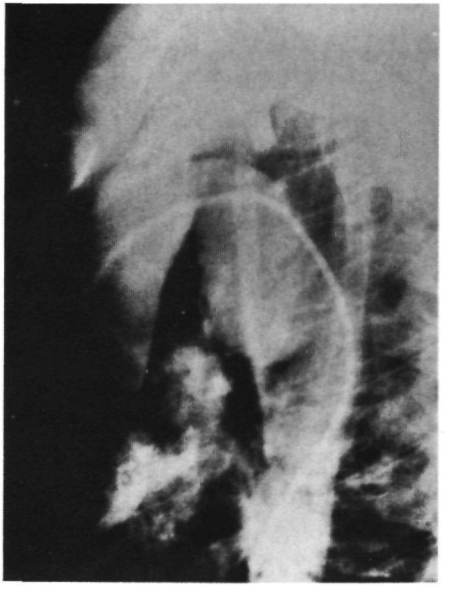

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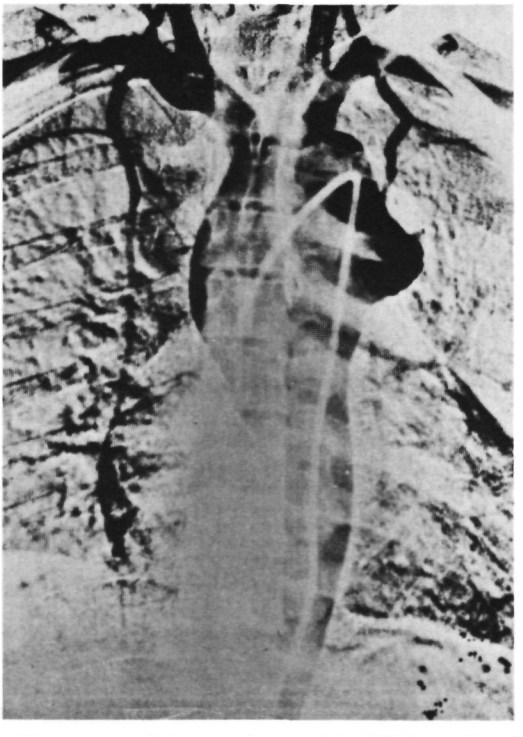

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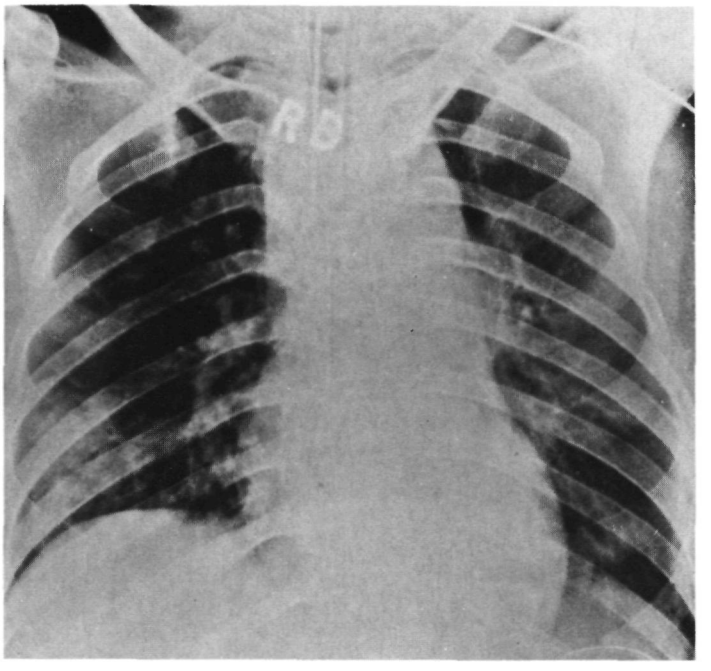
Figure VIII.3 Male, 24 years, car-driver, patient 5 with CTAR. a. Chest X-ray at the time of detection of the CTAR, 6 years after trauma: mass lesion with calcified rim at the level of the aortic knob. b. and c. AP and lateral angiography: false aneurysm at the level of the aortic isthmus.



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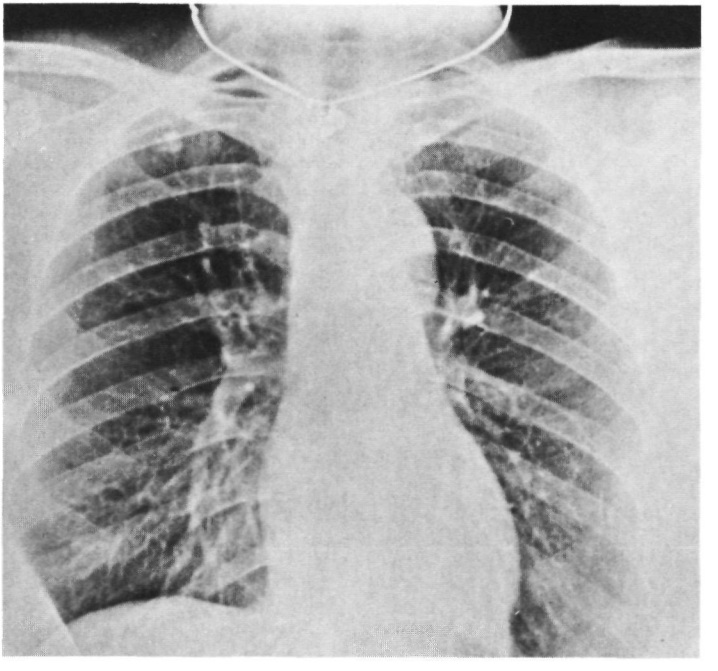
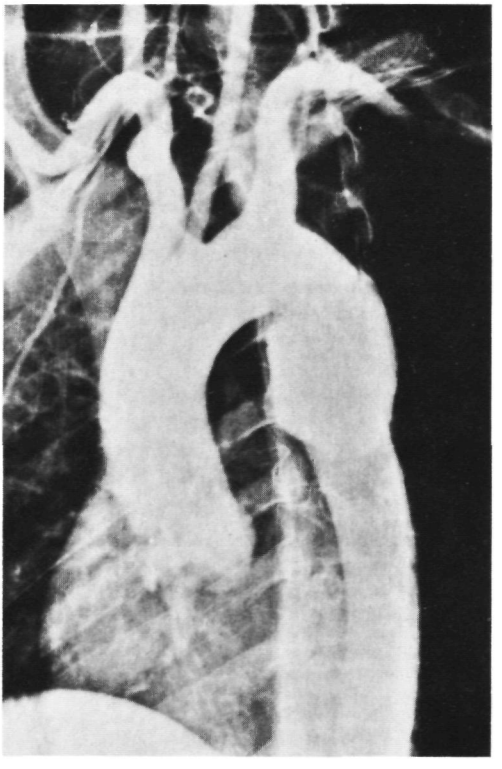
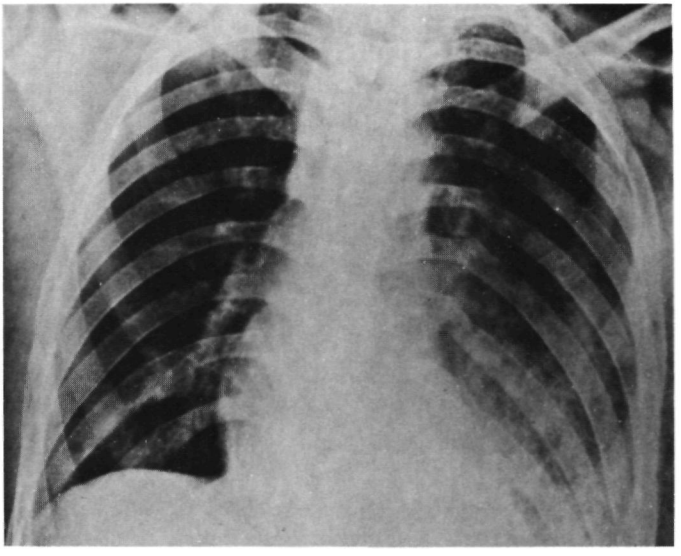


b

Figure VIII.4 Female, 26 years, car passenger, patient 7 with CTAR. a. Chest X-ray on admission 01.06.1976 : discrete widening of the mediastinum (7.5 cm). Normal position of the trachea and naso-gastric tube. Aortic knob not visible. Widened paratracheal stripe. Diagnosis of TAR missed.
b. Chest X-ray 31.08.1981: abnormal contour of aortic knob. c. Angiography: false aneurysm at the level of the aortic isthmus.





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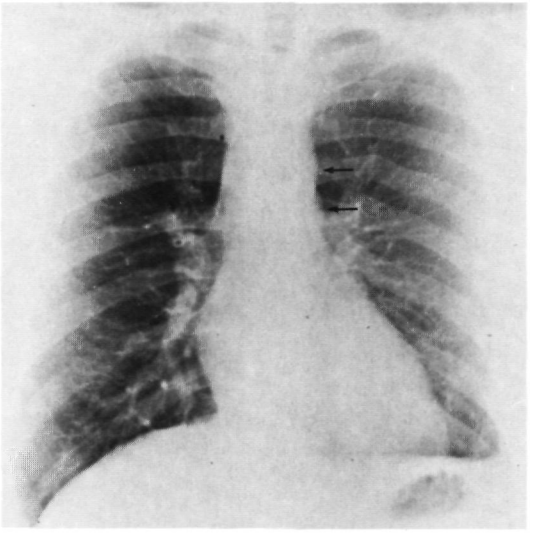
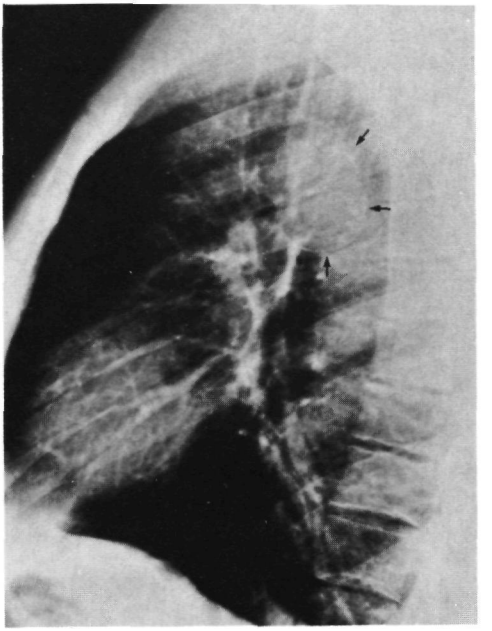


b

Figure VIII.5 Male, 18 years, motorbike-driver, patient 8 with CTAR. a. Chest X-ray on admission 19.07.1971: unsharp aortic outline; multiple rib fractures; apical cap. Contusion of left lower lung. (Underpenetrated low KV chest X-ray, evaluation of tracheal position not possible). Diagnosis of TAR missed. b. and c. Chest X-rays at the time of detection of CTAR (1981): tracheal displacement to the right; abnormal configuration below the aortic knob (arrows); calcified mass lesion on the lateral chest X-ray (arrows). d. Angiography: false aneurysm at the level of the aortic isthmus.



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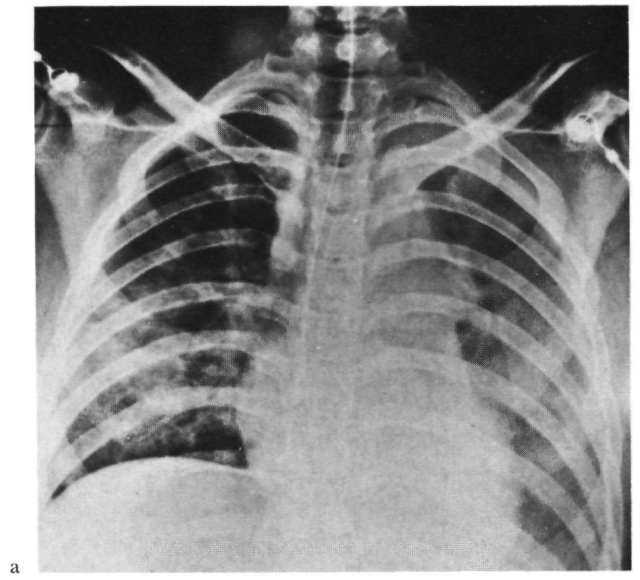

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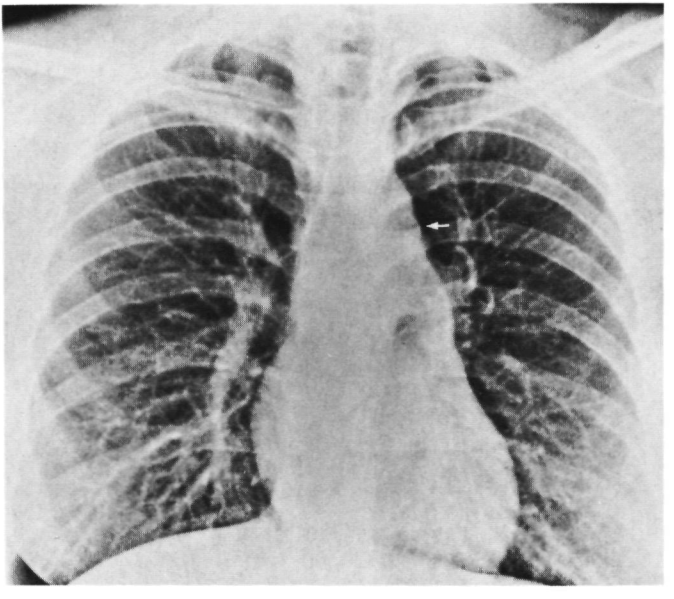

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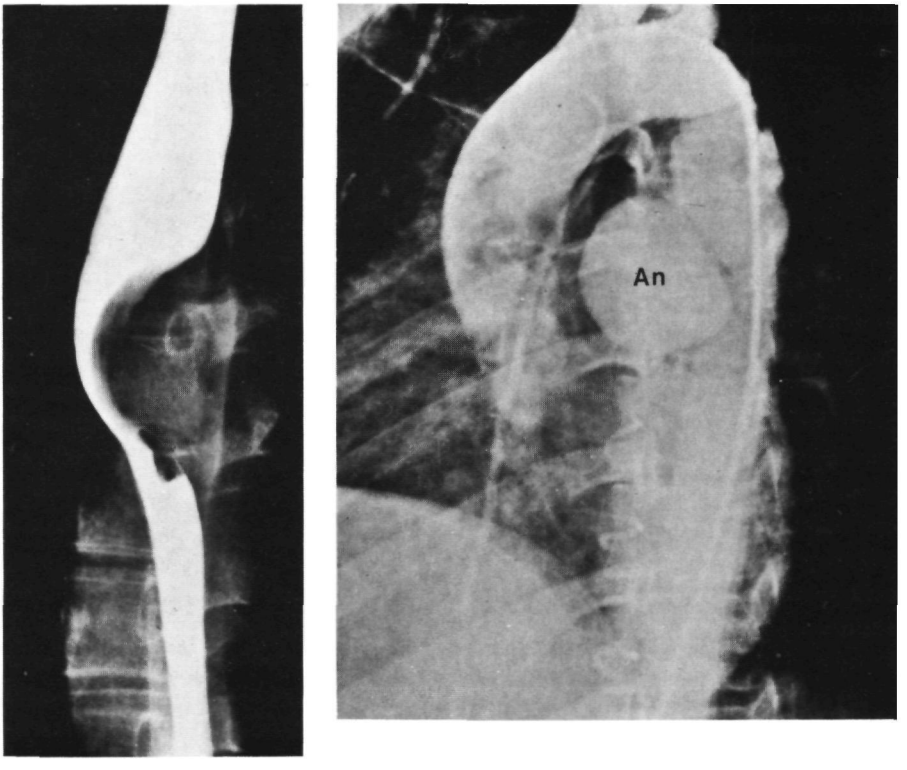
Figure VIII.6 Female, 21 years, car-driver, patient 10 with CTAR. a. Admission chest X-ray 20.07.1982 : widening of the mediastinum (7.5 cm); unsharp aortic outline; broadening of the right paratracheal stripe; displacement of the trachea to the right. Bilateral lung contusion. Diagnosis of TAR missed. b. Chest X-ray 25.11.1982 : abnormal contour of the aortic knob (arrow). c. X-esophagus: outside compression by mass lesion at the level of the aortic knob. d. Contrast enhanced CT scan, 'two chambered' false aneurysm at the level of the aortic isthmus. (An.: false aneurysm, Ao.: Aortic lumen). e. Angiography: medial and lateral localised false aneurysm below the level of the aortic isthmus.



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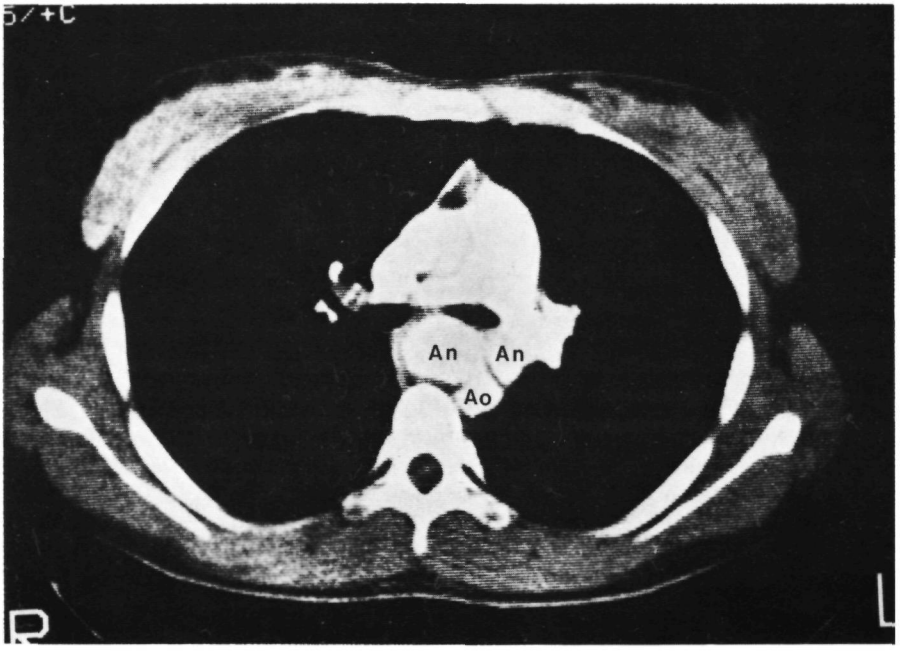

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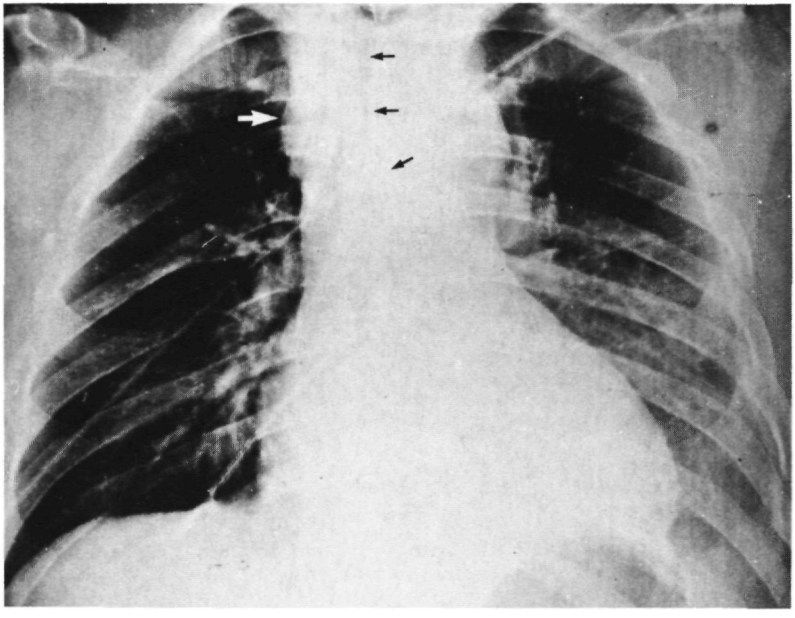

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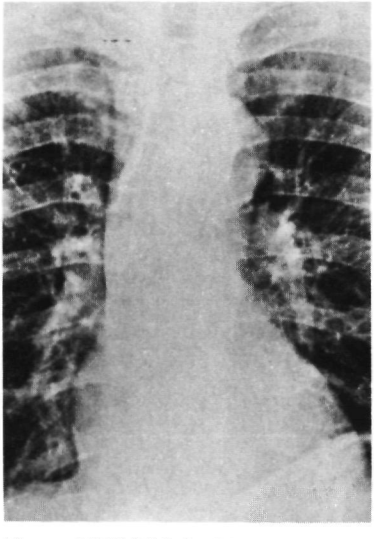

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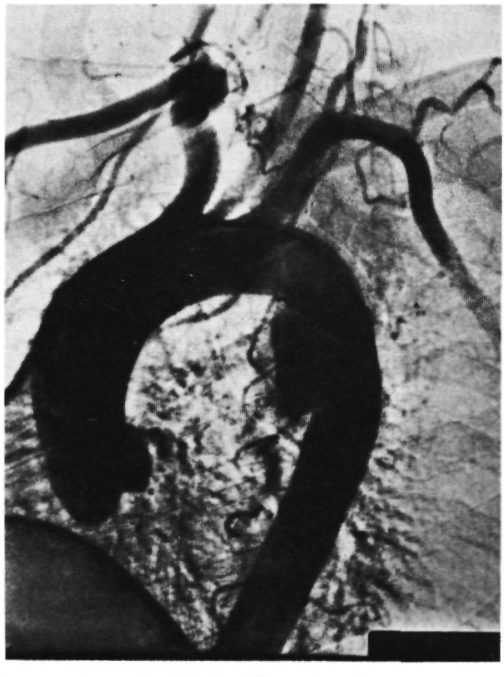

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Figure VIII.7 Male, 39 years, motor-driver, patient 11 with CTAR. a. Admission chest X-ray 25.07.1982: widening of the mediastinum (9.0 cm); unsharp aortic outline; widened paratracheal stripe (white arrow); displacement of the trachea (black arrows); multiple rib fractures. Diagnosis of TAR missed. b. Chest X-ray at second admission 25.01.1983: abnormal contour of the aortic knob. c. Angiography: false aneurysm of the level of the aortic isthmus.

Table VIII.5 Surgery in patients with chronic traumatic aneurysms: 10 patients operated upon of the 11

Patient nr.	*Tear circumference*	*Type protection*	*Surgery*	*Outcome*
1	2/3	LHB	Patch	Good
2	1/3	LHB	Graft	Good
3	1/3	LHB	Graft	Good
4	?	LHB	Graft	Good
5	Total	LHB	Graft	Good
6	Irresectable		–	Death
7	Total	No shunt	Graft	Good
8	1/3	No shunt	Graft	Good
9	No operation	–	–	Death
10	1/2	No shunt	Graft	Good
11	1/2	No shunt	Graft	Good

stenosis of the left main bronchus due to external compression. In another patient (patient 10) a CT scan examination of the thorax was performed (fig. VIII.6.d). A (false) aneurysm of the proximal descending aorta could be seen after i.v. contrast administration.

VIII.3.3. *Angiography*

The same technique in patients with CTAR was employed as described in chapter II.3.2. In one patient (patient 1), an image of the thoracic aorta was obtained by contrast injection in the central pulmonary artery. In the other 10 patients thoracic aortograms were performed via the femoral route. In one patient (patient 4), it was not possible to pass the false aneurysm from below and an additional angiogram via the axillary approach had to be performed to obtain information on the brachiocephalic vessels and aortic arch (fig. VIII.2.d). In all patients a false aneurysm with varying diameter at the isthmus was found. Most aneurysms were localised at the inner surface of the aortic arch with ventral extension. Most aneurysms showed delayed washout of the contrast medium. No abnormalities of the brachiocephalic vessels were discovered. No complications due to angiography were encountered.

VIII.4. **Surgery**

All patients except two were operated upon within weeks after the detection of the aneurysm. In one of these two patients there was a delay of two years - for unknown

reasons - before an angiogram of the thoracic aorta was obtained: the other patient died unexpectedly 36 hours after angiography. Operative findings, the type of shunt used, surgical therapy and the outcome are given in tabel VIII.5. There were no cases of postoperative paraplegia. One female patient (patient 3) sufferred from a severe pulmonary embolus on the 11th postoperative day, requiring median sternotomy and exploration of the pulmonary artery during extracorporal circulation. Ligation of the inferior vena cava was performed. She recovered despite anuria for which intermittent dialysis was performed.

VIII.5. **Survival**

One patient (patient 6) died during surgery because the false aneurysm had ruptured into the mediastinum and pericardium while crossclamping of the aorta was not possible. One patient died unexpectedly 36 hours after angiography and before operation due to acute rupture with perforation of the aneurysm into the left main bronchus and massive hemoptoe. All other patients (n = 9) had an uneventful recovery without postoperative complications, except the patient with the postoperative pulmonary embolism.
To date, no late complications have occurred in the operated patients.

VII.6. **Discussion**

It is difficult to explain why the diagnosis of TAR was missed during the initial hospitalisation of our patients in whom a chronic traumatic aneurysm was later discovered. There were many signs indicating mediastinal hematoma on their initial thoracic roentgenograms (table VIII.3). Probably one of the reasons is that until some years ago, the radiographic findings of TAR were less well known. Vascular injury may escape diagnosis, because rupture at the isthmus is associated with little external evidence of chest trauma in one third of the cases (Parmley et al., 1958).
In addition, orthopedic or neurosurgical problems may so dominate the clinical picture that the chest X-ray is not carefully evaluated (Schonholtz and Janhke, 1964; Jackson et al. , 1965). As is the case with acute TAR, radiography plays an essential role in detecting a chronic posttraumatic aneurysm. Sometimes findings are rather subtle and can easily be overlooked. The most important changes are contour deformities of the mediastinum at or below the aortic knob, sometimes with a calcified rim. Contrast examination of the esophagus can show a local impression by the aneurysm at the level of or below the aortic knob (case 3.10), (Fig. VIII.1.c and VIII.6.c). CT (Fig. VIII.6.d) can show the aneurysm after contrast administration, but in general the surgeon will need an angiogram of the aortic arch,

brachiocephalic vessels and the descending aorta. In these stable patients, who can cooperate well, there will be a defenite role for digital substraction angiography (DSA), either intravenous or intraarterial; by this relatively noninvasive method, all the information needed for the surgeon can be obtained.

It is clear from the literature and our patient data that an elective operation for a stable chronic traumatic aneurysm carries a low risk. It is known from surgery for abdominal aneurysms, that the mortality rate for imminent or complete ruptured aneurysms is much higher than when elective operation is possible. The same seems to hold for a growing thoracic aneurysm as was the case in patient nr. 6. The fatal outcome in case 9 underlines the unpredictable course of a long standing chronic traumatic aneurysm (12 years). Our patients showed on average a rather short time (5.3 years) between the initial trauma and the detection of the aneurysm. Despite this short time interval, five patients had more or less severe symptoms of thoracic pain, dysphagia, hoarseness and dyspnea. Because of the unpredictable course of a chronic traumatic aneurysm, the possible life-threatening complications (Bennett and Cherry, 1967), the low operative mortality, and the long life expectancy of these young patients, it seems advisable to operate upon these patients electively soon after detection of the aneurysm.
According to Parmley et al. (1958) and McBurney and Vaughan (1961), 2 - 5% of all acute traumatic aneurysms develop into a chronic aneurysm. We have seen 11 patients with a chronic traumatic aneurysm - this means that in the same period there should have been 400 - 800 cases in the two areas, from where patients are referred to both University Hospitals. With an initial mortality of 80% we should have seen 80 - 160 clinical patients. In reality we have seen 68 clinical cases with TAR. These data indicate that TAR is probably underdiagnosed on our areas.

In this thesis radiological and clinical aspects of acute and chronic traumatic rupture of the thoracic aorta are discussed on the basis of a study of the literature and of studies that were made in 57 patients with an acute aortic rupture and 11 patients with a chronic aneurysm of the thoracic aorta. The patients were referred to the University Hospitals in Nijmegen (n = 44) and in Groningen (n = 24) during the period 1970-1984. As a consequence of this extended time interval, some of the data could only be compiled retrospectively, with all the shortcomings inherent in such studies. On the other hand, most of the radiological investigations were supervised by the same radiologists. After an introduction and outline of the study (chapter I) the thesis is divided in two parts, part I (chapter II-VII) deals with the acute traumatic aortic rupture and part II (chapter VII and VIII) with the chronic traumatic aneurysm of the thoracic aorta.

Part I
ACUTE TRAUMATIC ANEURYSMS OF THE THORACIC AORTA

In chapter II, general aspects of acute traumatic ruptures are described on basis of a search of the literature. These aspects are: 'historical data', 'frequency of occurrence', 'ethiology and pathogenesis', 'pathology', 'natural history' and 'diagnosis'. Under the heading diagnosis, clinical symptoms, chest radiography, angiography, and the use of newer modalities such as digital subtraction angiography (DSA) and computer tomography (CT) are reviewed. It is to be expected that DSA will be of importance in the evaluation of TAR, especially in stable patients, however its proper role has still to be evaluated to fuller extend. The application of CT in the diagnosis of TAR will probably be limited. The chest X-ray still remains the best basic diagnostic tool.

In chapter III, data of the own patients (57 patients with TAR and data of the initial trauma of 6 patients with a CTAR; together 63 patients) with TAR are given with special attention to the kind of trauma and the time interval between trauma and diagnostic procedures. For comparison of the clinical and radiological findings of our patients with TAR we took data from a group of blunt chest trauma patients (n = 62) without - angiographically proven - traumatic aneurysm. Both groups were comparable as regard to age and kind of trauma. Most patients with TAR were involved in automobile accidents (52/63), although other trauma mechanisms were observed.

The most frequent clinical symptoms in the TAR patients were chest pain and dyspnea. From the comparison with the control patients, these appeared to be rather aspecific symptoms for TAR and could be attributed to fractures of the thoracic cage (n = 29) and/or pulmonary contusion (n = 22). Clinical symptoms specific for aortic rupture such as an acute coarctation syndrome (n = 5) or hoarseness (n = 1) were infrequently present.

The chest radiographs were evaluated for 15 signs, known to be related to hemomediastinum, in both groups.

The signs of mediastinal bleeding, which were significantly more frequent in patients with TAR than in the control patients included: displacement to the right of the trachea; displacement downward and to the right of the left main bronchus; displacement to the right of a nasogastric tube; unsharp aortic contours; an opacified pulmonary window; widened left and right paraspinal lines; and broadening of the right paratracheal line of more than 5 mm, (all signs $p < 0.01$). Displacement of the superior vena cava was also more frequent in the TAR group ($p < 0.05$).

Mediastinal widening of either > 8 cm or ≥ 10 cm was more frequent in the TAR group than in the control patients ($p < 0.05$). Mediastinal chest width ratios were relatively high in both groups and were not useful in differentiating the groups ($p > 0.05$).

The best objective signs to distinguish TAR in patients with hemomediastinum were an opacified pulmonary window, a broadened paratracheal stripe, a widened right paraspinal line and a displaced nasogastric tube.

The combination of an opacified pulmonary window, a widened paratracheal stripe and a widened right spinal line appeared to have the best positive correlation with TAR (100% ± 16%). This positive correlation was lower for all other combinations (51-78%).

As not always all of the above mentioned signs are present in patients with hemomediastinum, we estimate that by thoroughly analysing the chest X-rays of patients with blunt chest trauma, the number of negative angiograms can be reduced and one out of two or three will be positive for TAR, in stead of the former one out of five or six angiographies. One or more signs of hemomediastinum on the chest X-ray of a patient with blunt thoracic trauma dictates angiography of the thoracic aorta; some (combinations of) signs have a higher specificity and sensitivity for TAR compared with others. No sign has a sensitivity or specificity of 100%.

Angiography was performed on 52 patients as in two patients the diagnosis was missed, while three patients died before angiography could be performed. Angiographically the location of the aneurysm was in the isthmus area in 43 patients, in the aortic arch in four patients, in the descending aorta in five patients. One patient showed multiple aortic ruptures. The most frequent presentation was a false aneurysm, frank dissection was seen in two patients and occlusion of the aortic lumen in another two patients. Angiography was performed by the femoral pathway in 49 patients, by the brachial approach in one patient and in 2 patients both routes were

used. There were no complications from angiography. No patients were operated upon without angiography.

In chapter IV the concomitant traumata of patients with TAR are discussed. Most concomitant injuries were present in the CNS system, the liver, the spleen and the extremities. One patient died at surgery due to concomitant injuries to abdominal organs. Nine patients died postoperatively due to CNS injury or sequelae of abdominal or thoracic injuries.
These injuries were the reason for the substantial postoperative death rate. From the data of our patients could be concluded that severe non-thoracic concomitant injuries, especially of the liver and spleen, were more frequent in TAR patients than in control patients.

In chapter V surgery for patients with TAR is elucidated. A survey of the literature, reviewing surgical techniques, methods to prevent distal ischemia, other operations and complications is given.
Of our own patients with TAR, 46/57 underwent a thoracotomy for TAR. In most (n = 36) of our patients repair of the rupture was performed using a prosthetic dacron graft, with the help of a left heart bypass (n = 30) to prevent distal ischemia and systemic heparinisation. Eleven patients were operated upon with crossclamping of the aorta under pharmacological control of the systolic tension and without a shunt. Five patients were operated with the TDMAC shunt and without heparinisation. In four patients a direct repair of the aorta was possible and in two patients the aortic wall defect was repaired with a patch. Six patients died during the operation, all except one due to the aortic rupture. Of these six patients, two were unsalvageable because the rupture was located in the aortic arch and/or dissection had extended to the ascending aorta. Three patients died from TAR, one during a diaphragmatic repair, the other two were in a desperate clinical condition at the time of the operation; another during celiotomy from liver injury. Two patients died from hemorrhage directly related to systemic heparinisation, one patient peroperatively, the other the next day. Four patients developed a postoperative paraplegia, three of which had been operated without a shunt. In many instances (n = 29) two or more operations were performed; often (n = 17) a celiotomy was performed first because of frank bleeding or to prevent aggravated bleeding by heparinisation from abdominal injury.
Postoperative complications due to thoracic organ injury were present in 16 patients, due to other organ injury in 29 patients. In total 30/46 (65%) of our patients, who underwent repair of the aortic rupture, survived. From our results and from the literature it is not yet clear which kind of distal protection during operation is to be preferred, because on the one hand hemorrhage is a serious complication from systemic heparinisation and on the other hand the number of patients with postoperative paraplegia after simple crossclamping is high in our series.

In chapter VI the prognosis for TAR is discussed. The overall survival of the 57 patients in the acute group was 30/57 (53%). Survival after surgery for TAR was 30/46 (65%). Our experience is in agreement with the literature that TAR is always an unstable lesion: in two patients the diagnosis was missed and these two patients died within 24 hours; three patients died before angiography and four patients in the short time between angiography and operation. From our data and the literature it is evident that the time factor is of utmost importance in saving patients with TAR. Another important factor is the presence of concomitant injuries, especially of cerebral and abdominal injuries. No difference in survival was found in patients operated upon first for their abdominal injury compared with those who were operated upon first for their aortic rupture. 9/57 (16%) deaths were directly or indirectly related to concomitant CNS or abdominal organ injuries. Survival without concomitant injury(n = 4) was 100%.

Part II
CHRONIC TRAUMATIC ANEURYSMS OF THE THORACIC AORTA

Chapter VII analyses the literature of chronic traumatic aneurysms of the thoracic aorta, detailing in particular a survey of the natural history, pathology, diagnosis and surgery.

In chapter VIII eleven own patients are described who were referred to one of the University Hospitals 3 months to 12 years after the initial trauma. Four patients presented no symptoms at all, three patients showed slight or aspecific symptoms as thoracic pain, while the remaining four patients showed more severe complaints like dysphagia, hoarseness or dyspnea. In the group of patients with complaints, the aneurysm was detected because chest X-rays were ordered, in the other patients the aneurysm was detected by chance. In the retrospective evaluation of the available chest X-rays at the time of the initial admission, it was not clear why the TAR was not detected at that time: some signs on the chest roentgenograms were present that should have raised suspicion of TAR. At the time of detection of the CTAR, chest X-rays showed either an obvious mass at the region of the aortic knob or slight contour deformities at that level sometimes with a rim of calcification. In one patient, the aneurysm was irresectable at the time of operation because of its extension into the aortic arch: another patient died unexpectedly 36 hours after angiography. The other nine patients had an uneventful recovery and follow-up after operation.
Once a chronic posttraumatic aneurysm is detected, immediate repair seems advisable because of the unpredictable course and the low operative mortality.

In dit proefschrift worden de radiologische en klinische aspecten van acute en chronische traumatische rupturen van de thoracale aorta besproken aan de hand van de literatuur en studies die werden verricht bij 57 patienten met een acute ruptuur van de aorta en 11 patienten met een chronisch traumatisch aneurysma. De patienten werden verwezen naar het St. Radboud Ziekenhuis in Nijmegen (n = 44) en naar de Universiteitskliniek van Groningen (n = 24) in de periode 1970-1984. Tengevolge van dit lange tijdsbestek en het retrospectieve karakter van deze studie - met alle tekortkomingen van dien - waren niet van alle patienten de gegevens compleet. De meeste radiologische onderzoeken werden echter gesuperviseerd door dezelfde radiologen (de auteur van dit proefschrift te Nijmegen en Dr. L. Kingma te Groningen). Na de introduktie en het bespreken van het doel van de studie (hoofdstuk I), is dit proefschrift in 2 delen gesplitst: deel I (hoofdstuk II-VII) behandelt de acute traumatische rupturen, deel II (hoofdstuk VII en VIII) beschrijft de chronische traumatische aneurysmata.

Deel I
ACUTE TRAUMATISCHE RUPTUREN VAN DE THORACALE AORTA

In hoofdstuk II worden algemene aspecten van acute traumatische rupturen van de thoracale aorta besproken aan de hand van literatuuronderzoek. Deze aspecten zijn 'historische gegevens', 'frequentie van vóórkomen', 'etiologie en pathogenese', 'pathologie', 'natuurlijk verloop' en 'diagnostiek'. Onder het hoofd diagnostiek wordt de klinische symtomatologie beschreven evenals de radiodiagnostiek van de thorax, de angiografie en het gebruik van nieuwe onderzoekstechnieken zoals digitale subtractie angiografie (DSA) en computer tomografie (CT). Verwacht wordt dat DSA een rol zal gaan spelen bij de evaluatie van traumatische aorta rupturen, vooral bij stabiele patienten; de exacte rol van DSA by TAR zal echter nog verder moeten worden geevalueerd. De toepassing van CT bij de diagnostiek van traumatische aorta rupturen zal waarschijnlijk beperkt blijven. De gewone thoraxopname is nog steeds het basale diagnostisch instrument voor de diagnostiek van TAR.

In hoofdstuk III worden de gegevens van de eigen patienten met acute ruptuur (n = 63),(57 patienten met acute ruptuur en gegevens van het initieel trauma van 6 patienten met een chronisch aneurysma) gepresenteerd, o.a. de aard van het on-

geval en het tijdsinterval tussen trauma en diagnostiek. Als vergelijking fungeerden de klinische en radiologische gegevens van een groep patienten met een stomp thorax trauma zonder - angiografisch bewezen - aorta letsel (n = 62). Beide groepen waren vergelijkbaar qua leeftijdsopbouw en aard van het trauma. De meeste patienten met ruptuur waren betrokken bij een automobiel ongeval (n = 46), andere ongevalsmechanismen waren bij de andere patienten de oorzaak van de ruptuur. De meest frequente symptomen bij patienten met een acute ruptuur waren pijn op de thorax en dyspnoe, deze symptomen bleken bij vergelijking met patienten zonder ruptuur echter aspecifiek en konden worden toegeschreven aan fracturen van het thoraxskelet (n = 29) en long contusie (n = 22). Klinische symptomen specifiek voor aorta ruptuur zoals een acuut coarctatie syndroom (n = 5) of heesheid waren zelden aanwezig.

De thoraxopnamen werden beoordeeld op de aanwezigheid van haemomediastinum volgens 15 in de literatuur gehanteerde criteria. Kenmerken van mediastinale bloeding die significant frequenter voorkwamen bij patienten met een traumatische aorta ruptuur waren: verplaatsing naar rechts van de trachea; verplaatsing naar rechts en naar beneden van de linker hoofdbronchus; verplaatsing naar rechts van een maagslang; onscherpe contouren van de aorta; opgevuld aorto-pulmonaal venster; verbrede rechter en linker paraspinale lijn; verbreding van de paratracheale 'stripe' van meer dan 5 mm,(alle kenmerken $p<0.01$). Verplaatsing van de vena cava was eveneens meer frequent in de ruptuur groep ($p<0.05$). Zowel mediastinum verbreding van 8 cm of meer of van 10 cm en meer, kwamen frequenter voor bij de patiënten met een ruptuur dan in de controle groep ($p<0.05$). De ratio thorax-/mediastinum breedte was relatief hoog in beide groepen en was niet bruikbaar om beide groepen te differentieren ($p>0.05$).

Indien een of meer tekenen van haemomediastinum aanwezig zijn op de thoraxopname, dient een angiografie plaats te vinden om een ruptuur uit te sluiten, cq aan te tonen. Sommige (combinaties van) kenmerken bezitten een grotere sensitiviteit of specificiteit voor aorta ruptuur dan andere. Geen enkel kenmerk bleek 100% sensitiviteit of specificiteit te bezitten voor aortaruptuur.

De beste objective discriminerende kenmerken voor traumatische aorta ruptuur in de patienten met een haemomediastinum waren: een opgevuld aorto-pulmonaal venster; een verbrede paratracheale lijn; een verbrede rechter paraspinale lijn en een verplaatste oesophagus sonde. De combinatie opgevuld aorto-pulmonaal venster, een verbrede paratracheale en een verbrede paraspinale lijn bleken de beste positieve correlatie te bezitten met een traumatische aorta ruptuur (100% $\pm$ 16.8%). Deze positieve correlatie was kleiner voor alle andere combinaties (51-78%). Omdat niet bij alle patienten met een traumatische aortaruptuur bovenvermelde kenmerken aanwezig zijn of geevalueerd kunnen worden, schatten wij de kans op een positieve angiografie (dwz aangetoonde aorta ruptuur) bij patienten met een traumatisch haemomediastinum als één op twee of drie angiogrammen. Dit betekent een reductie van het aantal negatieve angiogrammen, daar voorheen

slechts ongeveer een op 5 of 6 angiogrammen bij onze patienten met stomp thorax trauma positief was.
Angiografie werd verricht bij 52 patienten - drie patienten overleden voordat een angiografie kon worden verricht; bij twee patienten werd de diagnose gemist - en daarbij bleek de aorta ruptuur gelokaliseerd in het isthmusgebied bij 43 patienten, in de aortaboog bij vier patienten en in de aorta descendens bij vijf patienten. Bij één patient werden multipele rupturen aangetoond. Meestal was een vals aneurysma aanwezig, dissectie werd bij twee patienten gezien, terwijl occlusie van de aorta bij twee patienten werd aangetoond. Complicaties tengevolge van de angiografie traden niet op; zonder angiografie werden geen patienten geopereerd. Angiografie via de arteria femoralis werd verricht bij 49 patienten. Bij een patient werd via de arteria brachialis arteriografie verricht en bij 2 patienten werden zowel de femorale als de brachiale route gebruikt.

In hoofdstuk IV worden de bijkomende traumata van patienten met acute ruptuur besproken. De meeste begeleidende traumata betroffen het centraal zenuwstelsel, de lever, de milt en de extremiteiten.
Een patient overleed peroperatief tengevolge van letsels van de lever en milt. Negen patienten overleden postoperatief tengevolge van cerebrale letsels of tengevolge van abdominale of thoracale letsels. Deze letsels waren dus een belangrijke oorzaak van postoperatieve letaliteit. Uit onze gegevens bleek dat de begeleidende letsels, vooral van andere organen dan de thorax, vaker voorkwamen bij patienten met acute ruptuur dan in de controlegroep.

In hoofdstuk V wordt de chirurgische behandeling van patienten met aorta ruptuur besproken. Na een bespreking van de literatuur over de chirurgische techniek en de methoden om distale ischaemie te voorkomen, worden andere operaties en complicaties beschreven.
Van de 57 patienten met aorta ruptuur ondergingen er 46 een thoracotomie met als doel herstel van de aorta ruptuur. Bij de meeste patienten (n = 36) werd de ruptuur hersteld door middel van een dacron prothese met behulp van een links bypass (n = 30) om distale ischemie te voorkomen, gecombineerd met systeem heparinisatie. Elf patienten werden geopereerd met alleen afklemmen van de aorta gecombineerd met farmacologische beinvloeding van de systolische druk; vijf patienten werden geopereerd met behulp van de TDMAC shunt zonder heparinisatie.
Slechts bij vier patienten was een directe anastomosering mogelijk en bij twee patienten werd het defect in de aortawand hersteld met een patch of overhechting.
Zes patienten overleden peroperatief, allen behalve één tengevolge van de aorta ruptuur. Van deze zes patienten waren er twee 'inoperabel' omdat de ruptuur in de aortaboog was gelokaliseerd of omdat de ruptuur zich tijdens operatie uitbreidde naar de aorta ascendens. Drie patienten overleden tengevolge van de ruptuur; een terwijl herstel van een diafragma ruptuur werd uitgevoerd, de andere twee bevon-

den zich in een zeer slechte klinische conditie ten tijde van de operatie. Een patient overleed tijdens laparotomie aan verbloeding tengevolge van leverletsel. Twee patienten overleden postoperatief tengevolge van verbloeding die verband hield met de systeem heparinisatie. Vier patienten ontwikkelden een postoperatieve paraplegie, van deze vier patienten waren er drie geopereerd met alleen afklemming van de aorta, zonder shunt. Bij veel patienten (n = 29) werden twee of meer operaties uitgevoerd; vaak (n = 17) werd een laparotomie als eerste operatie uitgevoerd. De reden hiervan was dat bloeding tengevolge van abdominaal letsel aanwezig was. Postoperatieve complicaties van de thoracale organen traden bij 16 patienten op; in andere organen bij 29 patienten. De postoperatieve overleving was na operatie voor ruptuur 30/46 (65%). Noch uit onze resultaten en evenmin uit de literatuur is duidelijkheid te verkrijgen over de ideale methode om distale ischemie te voorkomen: enerzijds is bloeding die kan optreden tengevolge van heparinisatie een ernstige complicatie, anderzijds vonden wij een relatief hoog aantal patienten met postoperatieve paraplegie bij die operaties waarbij alleen afklemming werd gebruikt (3/11).

In hoofdstuk VI wordt de prognose van onze patienten met acute aorta ruptuur besproken. De overleving van de totale groep met acute ruptuur was 30/57 (53%). Overleving in de groep die geopereerd werd voor aorta ruptuur bedroeg 30/46 (65%). Onze ervaringen dat acute aorta rupturen onstabiele letsels zijn komen overeen met de literatuur. Bij twee patienten werd de diagnose gemist, beide overleden binnen 24 uur. Drie patienten overleden voordat angiografie kon worden uitgevoerd en vier patienten overleden in de korte tijd tussen angiografie en operatie. Uit onze bevindingen blijkt dat de tijdsfactor van groot belang is voor de overleving van patienten met een aorta ruptuur. Een andere factor van grote betekenis voor de overleving is de aanwezigheid van bijkomende letsels, vooral cerebrale en abdominale letsels. Er bleek geen verschil in overleving te bestaan tussen patienten die eerst thoracaal (n = 28) of eerst abdominaal (n = 17) geopereerd waren. Bij 9 van de 57 patienten (16%) stond de doodsoorzaak direct of indirect in relatie met begeleidende letsels van de hersenen of buikorganen. Overleving zonder begeleidende letsels was 100%, het betrof hier echter slechts vier patienten.

Deel II
CHRONISCHE TRAUMATISCHE ANEURYSMATA VAN DE THORACALE AORTA

Hoofdstuk VII behandelt de literatuur van de chronische traumatische aneurysmata van de thoracale aorta, met name een overzicht betreffende natuurlijk beloop, pathologie, diagnostiek en chirurgie.

In Hoofdstuk VIII worden elf patienten besproken die naar een van de Universiteitsklinieken (Nijmegen n = 9, Groningen n = 2), drie maanden tot 12 jaar na het initiele trauma werden verwezen. Vier patiënten vertoonden geen klachten, drie patienten vertoonden lichte of aspecifieke klachten, terwijl bij vier patienten min of meer ernstige klachten aanwezig waren zoals dysphagie, heesheid of dyspnoe. Naar aanleiding van de klachten werd röntgenonderzoek van de thorax en/of oesophagus verricht en hierop werd het aneurysma ontdekt; bij de patiënten zonder klachten werd het aneurysma bij toeval (screening) aangetoond.
Bij de retrospectieve evaluatie van de beschikbare thoraxfoto's ten tijde van de eerste opname (n = 5) werd het niet duidelijk waarom het aneurysma ten tijde van het initieel trauma niet was aangetoond; er waren verschillende aanwijzingen aanwezig - hoewel soms subtiel - die wezen op het bestaan van een haemomediastinum. Ten tijde van ontdekking van het chronisch aneurysma liet de thoraxfoto een weke delen zwelling zien ter hoogte van de aortaknop of subtiele contourveranderingen ter hoogte van de aortaknop, soms samen met een kalkschilletje.
Bij operatie van het aneurysma, bleek dit bij één patient irresectabel omdat er een uitbreiding bestond naar de aortaboog; een patient overleed onverwacht 36 uur na de angiografie. Alle andere 9 patienten herstelden voorspoedig zonder late complicaties.
Het lijkt wenselijk een eenmaal ontdekt chronisch traumatisch aneurysma te opereren vanwege het onvoorspelbare verloop en de lage operatieve mortaliteit.

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V *Kodeerformulieren Aorta-rupturen*

Naam
Plaats ongeval
1 Volgnr
2 Geb datum
3 Geslacht 1 = man
2 = Vrouw
4 Leeftijd 0 = 0-9
1 = 10-19
2 = 20-29
3 = 30-39
4 = 40-49
5 = 50-59
6 = 60
5 *Ziekenhuis*
1 = Radboud zhs Nijmegen
2 = Acad zhs Groningen
Datum + tijd opname
6 ZHS 1 = dat
7 ZHS 2 = dat
8 *Aard trauma*
1 = Horizontale deceleratie
2 = Verticale deceleratie
3 = Combinatie 1 + 2
4 = Anders
9 *Patient*
1 = Auto (bestuurder)
2 = Autopassagier
3 = Fietser
4 = Bromfietser
5 = Motorrijder/passagier
6 = Piloot/passagier
7 = Voetganger
9 = Andere
10 *Tijdsverloop trauma* E-H
1 = 0- 1 uur
2 = 1- 3 uur
3 = 3- 6 uur
4 = 6 uur
5 = 24 uur
6 = onbekend
11 Tijdsverloop trauma X-thorax
12 Tijdsverloop trauma-angiografie
13 Tijdsverloop trauma-operatie
Klinische bevindingen evt wijzend op aorta-ruptuur
15 Dyspnoe
16 Afname pulsaties arm re
17 Afname pulsaties arm li
18 Acuut coarctatie syndroom
19 Anurie
20 Paraplegie
21 Systolisch geruis
Thorax
22 1 = AP liggend
2 = PA staand
3 = Comb 1+2
4 = Lat opname
23 Thorax 1 = Symmetrisch
2 = Asymmetrisch
24 Adipositas pat
Thorax bevinding
25 1 = mediast 6- 8 cm
2 = mediast 8-10 cm
3 = mediast > 10 cm
4 = mediast < 6 cm
26 MC I
27 MC II
28 MC III
29 Trachea verplaatst naar re
30 Verplaatste li hoofd bronchus naar caudaal
31 Verplaatste oes naar re

32 Verplaatste Vena Cava naar re.
33 Onscherpe Aortaboog contour
34 Onscherpe contour
Aorta desc.
35 Opacific.Clear Space
tussen aorta en A. pulm.
36 Apical cap li.
37 Vage longbegrenzing
li. boven
38 Haemothorax li.
39 Verbrede paraspin.lijn li.
40 Verbrede paraspin.lijn re.
41 le of 2e ribfract.
42 Mult. ribfract. plus le of 2e
43 Andere ribfract. dan le of 2e
44 Vervolg thorax aanwezig
45 Verander. thorax in tijd
46 *Andere afw. op thorax*
47 Longcontusie
48 Diafragma-rupt.(verdenking)
49 Pneumothorax
50 Sternum fractuur
51 Andere afw. op thorax
52 *Andere Traumat. afw.*
53 Nierruptuur
54 Extremit. fracturen (mult.)
55 Leverruptuur
56 Miltruptuur
57 Diafragma-ruptuur
58 Perforatie darm
59 Epi/subdur. Haematoom
60 Wk fracturen (thorac)
61 *Arcografie*
0 = geen
1 = femoraal
2 = brachiaal
62 Abdomin. aortogr.
63 Complicatie angio
64 *Bevinding angio*
1 = Normaal
2 = Vals aneurysma
2 = Dissectie
4 = Intima scheur
5 = Transectie
6 = Extravasatie contrast
7 = Ductus divertikel
8 = Mult. aneurysmata
9 = Andere
66 *Laesie brachiocephale vaten*
1 = Aneurysma
2 = Dissectie
3 = Combinatie 1 + 2
4 = Geen
66 *Localisatie aortaruptuur*
1 = Isthmus
2 = Aorta asc.
3 = Aortaboog
4 = Aorta desc.
5 = Andere locatie
6 = Geen
67 *Chirurgische bevindingen*
1 = Normaal
2 = Vals aneurysma
3 = Dissectie
4 = Intima scheur
5 = Transectie
6 = Multipele aneurysmata
7 = Andere
68 *Bloeding*
1 = Mediastinum
2 = Pleura
3 = Geen
69 *Chirurgische therapie*
1 = Resectie + End to End anastomose
2 = Graft
3 = Andere
4 = Geen
70 *Chirurgische techniek*
1 = Li. atrium - Fem. bypass
2 = Crawford
3 = TMDAC shunt
4 = Andere
5 = Geen
71 *Operatie:*
1 = Alleen thorac.
2 = Thor. + abdomen
3 = Thor. + cerebr.
4 = Thor. + extrem.
5 = Thor. + abd. + extrem
6 = Thor. + abd. + cerebr.

7 = Thor. + abd. + cerebr. + extrem.

72 *Eerste operatie*
1 = Thoracaal
3 = Cerebraal
4 = Extremiteiten

73 *Beloop*
1 = Herstel
2 = Overl. vóór angio
3 = Overl. na angio vóór operatie
4 = Overl. tijdens operatie
5 = Overl. na operatie
6 = Overl. geen angio – geen operatie

74 *Postoperatie complic. thorac.*
1 = RDS
2 = Infarct
3 = Longembolie
4 = Andere
5 = Geen

75 *Andere postoper. complicaties*
1 = Abdominaal
2 = Cerebraal
3 = Paraplegie
4 = Anurie
5 = Sepsis
6 = Geen
7 = Andere

76 Overleden 1 = ja
2 = nee

77 Obductie

78 *Obductie bevindingen*
1 = Ruptuur
2 = Andere bevindingen

79 Datum ontslag

80 *Datum + tijd overlijden*

Tijdsduur operatie

81

82

83 *Cardiale pathol.*

84 Trachea verplaatsing naar ventr.

85 *Paratracheale stripe*
1 = 5mm
2 = 5mm

86 Grootte ruptuur
1 = 1/4
2 = 1/2
3 = 1/4
4 = volledig
5 = geen

APPENDIX B

Statistics

The data of the TAR and control group were compared with each other using the chi-square test. P values are given for testing the hypothesis that there are no differences between both groups. (for V numbers: see items on the purpose written computer program; Appendix A).

Variable	*P*	*Variable*	*P*	*Variable*	*P*
V25	<0.05	V34	<0.01	V49	<0.05
V29	<0.01	V35	<0.01	V51	<0.05
V30	<0.01	V39	<0.01	V55	<0.05
V31	<0.01	V40	<0.01	V56	<0.05
V32	<0.05	V41	<0.01	V57	<0.05
V33	<0.01	V42	<0.05	V85	<0.01

For the other variables (V14, V15, V26 to V60) $P>0.05$.

A stepwise discriminant analysis was performed (SAS program stepwise) to select potential discriminating variables. The most important were: V30, V31, V32, V38, V40, V85.
V30 and V32 were eliminated because these are variables with a subjective character. With the help of combinations of these variables the posteriori probability can be calculated of being in the TAR or control group. The a priori probability for TAR of patients undergoing angiography was set at 20%. Using Bayes rule, we calculated the a posteriori probability of TAR, making double or triple combinations of V31, V35, V38, V40 and V85.
The a posteriori probability of no rupture is 100% minus the above calculated probability in the different combinations.

A posteriori probability of TAR for two variables

V35[2]	*V85*[4]	*TAR* *n(%)*	*Controls* *n(%)*	*A posteriori probability of TAR with standard error*	
0	0	0 (0)	13 (39.4)	0 %	(1.2)
0	1	1 (1.9)	5 (15.2)	3.1%	(3.2)
1	0	8 (15.4)	11 (33.3)	10.3%	(3.8)
1	1	43 (82.7)	4 (12.1)	63.0%	(11.0)
		52 (100)	33 (100)		
V35	*V40*[3]				
0	0	2 (4.3)	26 (52.0)	2.0%	(1.4)
0	1	0 (0)	0 (0)	–	
1	0	20 (43.5)	21 (42.0)	20.6%	(3.9)
1	1	24 (52.2)	3 (6.0)	68.5%	(12.5)
		46 (100)	50 (100)		
V85	*V40*				
0	0	5 (11.9)	21 (67.8)	4.2%	(1.8)
0	0	2 (4.8)	1 (3.2)	27.0%	(23.7)
1	0	15 (35.7)	8 (25.8)	25.7%	(7.0)
1	1	20 (47.6)	1 (3.2)	78.7%	(16.7)
		42 (100)	31 (100)		
V31[1]	*V85*				
0	0	0 (0)	11 (61.0)	0 %	(1.6)
0	1	9 (36.0)	5 (27.8)	24.5%	(8.6)
1	0	1 (4.0)	1 (5.6)	15.3%	(17.8)
1	1	15 (60.0)	1 (5.6)	73.0%	(19.4)
		25	18		

1) V31 Displaced nasogastric tube
2) V35 Opacified pulmonary window
3) V40 Broadened right paraspinal line
4) V85 Widened paratracheal stripe

A posteriori probability of TAR for three variables

V35[1]	*V85*[4]	*V40*[3]	*TAR* *n(%)*	*Controls* *n(%)*	*A posteriori probability of TAR with standard error*	
0	0	0	0 (0)	12 (40)	0.0%	(1.5)
0	0	1	0 (0)	0 (0)	–	
0	1	0	1 (2.4)	5 (16.7)	3.5%	(3.6)
1	0	0	4 (9.8)	9 (30.0)	7.5%	(3.8)
0	1	1	0 (0)	0 (0)	0.0%	
1	0	1	2 (4.9)	1 (3.3)	26.8%	(23.6)
1	1	0	14 (34.1)	3 (10)	46.1%	(14.6)
1	1	1	20 (48.8)	0 (0)	100 %	(16.8)
			41 (100)	30 (100)		
V35	*V85*	*V38*[2]				
0	0	0	0 (0)	12 (31.4)	0.0%	(1.3)
0	0	1	0 (0)	1 (3.0)	0.0%	(16.7)
0	1	0	1 (2.0)	5 (15.1)	3.1%	(3.3)
1	0	0	6 (11.7)	5 (15.1)	16.3%	(7.7)
0	1	1	0 (0)	0 (0)		
1	0	1	2 (3.9)	6 (18.2)	5.1%	(3.8)
1	1	0	29 (56.9)	2 (6.1)	70.1%	(14.6)
1	1	1	13 (25.5)	2 (6.1)	51.3%	(18.1)
			51 (100)	33 (100)		

1) V35 Opacified pulmonary window
2) V38 Left hemothorax
3) V40 Broadened right paraspinal line
4) V85 Widened paratracheal stripe

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CURRICULUM VITAE

De auteur werd geboren op 9 september 1944 te Bergen op Zoom. In 1963 – na het eindexamen Gymnasium β – begon hij met zijn studie geneeskunde aan de Katholieke Universiteit te Nijmegen. In 1969 werd het doctoraal examen en in 1971 het artsexamen behaald. De militaire dienst werd vervuld op de afdeling Radiologie van het Militair Hospitaal 'Dr. A. Matthijsen' (Hoofd: A. v.d. Beek).
Op 1 oktober 1972 begon hij zijn opleiding radiodiagnostiek in het St. Radboud Ziekenhuis te Nijmegen (Hoofd: Prof. Dr. Wm. Penn).
Na zijn inschrijving in het specialistenregister per 1 oktober 1976, is hij als staflid verbonden aan het Instituut voor Radiodiagnostiek van het St. Radboud Ziekenhuis (Hoofd tot 1982: Prof. Dr. Wm. Penn; thans: Prof. Dr. J.H.J. Ruys). Hij is verantwoordelijk voor de cardiale, vasculaire en pulmonale radiodiagnostiek.
De auteur is sinds 1970 gehuwd met Marie-José Lodewick en heeft twee kinderen, Susanne en Marjolein.

STELLINGEN

1. Klinisch wordt de diagnose van een traumatische aortaruptuur zelden gesteld; de thoraxopname is het belangrijkste instrument voor de diagnose.

2. Bij iedere patient met een stomp thorax trauma, gepaard gaande met deceleratie, dient de thoraxopname ook nauwkeurig beoordeeld te worden op kenmerken van haemomediastinum.

3. Indien een of meer kenmerken van haemomediastinum op de thoraxopname aanwezig zijn, is het gewenst een angiografie van de aorta te verrichten om een aortaruptuur uit te sluiten.

4. Verplaatsing van een oesophagus sonde, een opgevuld aorto-pulmonaal venster, een verbrede paratracheale stripe, en verbrede paraspinale lijnen zijn de beste discriminerende kenmerken voor patienten met een traumatische aortaruptuur.

5. De kans op een traumatische aortaruptuur is zeer klein indien het mediastinum normaal geconfigureerd is of indien de aortacontouren op de thoraxopname scherp afgrensbaar zijn.

6. Angiografie is een veilige procedure bij patienten met een traumatische aortaruptuur en moet bij iedere patient verdacht van ruptuur worden verricht.

7. Een vrij groot aantal (1 op 2 à 3) negatieve angiogrammen bij de diagnostiek van traumatische aortaruptuur moet als standaard geaccepteerd worden.

8. Patienten met een chronisch posttraumatisch thoracaal aneurysma hebben een betere long-term prognose als ze hiervoor geopereerd worden.

9. Het gebruik van laag osmolaire contrastmiddelen bij de bekken-been angiografie biedt weinig voordelen vergeleken met conventionele methyl-glucamine contrastmiddelen waaraan lidocaine is toegevoegd.
   Heystraten FMJ, Berg FG v. de, Mulderije ED. Diagnostic Imaging *52*: 141-144, 1983.

10. Biplane coronair installaties zijn een dure doch prettige luxe, vooral bij coronair dilatatie procedures.

11. De arteriele digitale substractie angiografie zal de conventionele platenangiografie in zijn geheel gaan vervangen.

12. Routine preoperatieve thoraxopnamen zonder anamnestisch of klinisch gegeven indicatie zijn overbodig.
Rucker L, Frye EB, Staten MA. JAMA *200*: 3209-3211, 1983.

13. Het is gewenst - gezien de digitale ontwikkeling in de radiologie - informatica en basale computerkunde in te bouwen in de opleiding van de radiologische laboranten.

14. Als reconstructie van een haemodynamisch actieve stenose van de arteria iliaca wordt overwogen, dan verdient transluminale dilatatie volgens Dotter de voorkeur.
Andel GJ v. Radiology *135*: 607-611, 1980.

15. De retrospectoscoop is niet alleen een van de gevoeligste maar ook een van de leerzaamste instrumenten in het armentarium van de radiodiagnost.

16. De (aorta) boog kan niet te lang gespannen staan.

6 december 1984 FMJ Heystraten